7. HUMAN EXPOSURE TO PARTICULATE MATTER: RELATIONS TO AMBIENT AND INDOOR CONCENTRATIONS

7.1 INTRODUCTION

The 1982 Air Quality Criteria Document for Particulate Matter and Sulfur Oxides (U.S. Environmental Protection Agency, 1982) thoroughly reviewed the PM exposure literature through 1981. The later "Second Addendum to Air Quality Criteria for Particulate Matter and Sulfur Oxides (1982)" (U.S. Environmental Protection Agency, 1986a) added coverage of newly available health effects information up to 1986. This chapter first summarizes key points from the 1982 Criteria Document, and then thoroughly reviews the PM exposure literature from 1982 through 1995 and includes some literature published and in press through February, 1996.

The U.S. Environmental Protection Agency (U.S. EPA) regulatory authority for PM only extends to the ambient air, defined in 40 CFR 50.1(e) as that portion of the atmosphere, external to buildings, to which the general public has access (Code of Federal Regulations, 1994). By the operative definition of ambient air, polluted air inside a building, or on private property owned or controlled by the source of pollution, is not regulated by the National Ambient Air Quality Standards (Costle, 1980; Bennett, 1983). However, it is necessary to consider total personal exposure to PM, both from the regulated ambient air and non-regulated indoor air. This is because ambient (outdoor) particles penetrate into non-ambient environments (indoors) where people spend approximately 85% of their time (U.S. Environmental Protection Agency, 1989). Therefore, when people are indoors, they are exposed to a mixture of ambient PM and particles generated indoors from non-regulated sources, such as PM from cigarette smoke and personal activities.

Personal exposure to total PM is important in itself, because the body may react differently to ambient and non-ambient particles of identical size but different chemical composition. Comparison of personal exposures to indoor and outdoor concentrations may provide clues as to whether or not these two types of PM have similar toxicity on a unit size and mass basis. Personal exposure may also act as a confounder in epidemiological studies which use an inferred community exposure to ambient PM as a parameter to correlate with community health

parameters, and an individual's personal exposure to total PM is a critical parameter for analysis if that person is a member of a cohort whose health outcomes are being tracked individually. Therefore, this chapter examines not only indoor air quality in regard to PM, but also community and individual exposures to PM, which include that portion of ambient PM which penetrates into indoor microenvironments (μEs). This is to aid in interpretation of acute and chronic epidemiology studies assessed in Chapter 12, in which ambient PM concentrations are assumed to be an indicator or a surrogate for mean community exposure to ambient PM or an individual exposure to ambient PM. Thus, this chapter has three objectives: (a) to provide a review of pertinent studies of indoor and personal exposures to PM; (b) to evaluate linkages between monitored personal exposures and exposures estimated from a fixed-site monitor located at some central monitoring site; and (c) to quantify the contribution of ambient air to personal PM exposure.

In this chapter, Sections 7.1.1 - 7.1.3 discuss the concept of ambient PM as a surrogate for a personal exposure and the relationship of a measured personal PM exposure to the ambient and nonambient concentrations of PM that may influence it.

Section 7.2 next reviews PM concentrations found indoors where people spend about 85% of their time (U.S. Environmental Protection Agency, 1989). This subject is discussed in detail because of the importance of indoor conditions for understanding total exposure to PM. Indoor air particles from indoor sources may be an important factor in the analysis and interpretation of epidemiology studies, because they may influence both the personal PM exposure and personal health of the exposed people.

Section 7.2.5 reviews the literature covering biological aerosols, which may produce direct health effects or act as a source of antigens capable of sensitizing people to the effects of other PM exposures.

Section 7.3 reviews the fundamental principles of personal PM monitoring and factors that influence the personal PM measurement.

Section 7.4 covers the literature on direct measurements of personal exposures to PM and PM constituents such as sulfates.

Section 7.5 reviews the literature on indirect exposure estimation procedures that predict exposures from time-weighted averages of concentrations measured indoors and outdoors.

Section 7.6 discusses the relationship of individual PM exposures to ambient PM concentrations and establishes a linkage between average personal PM exposures in a community to the ambient PM concentrations.

Section 7.7 discusses implications of PM exposure relationships for mortality and morbidity analyses.

Section 7.8 provides a Summary of Conclusions for Chapter 7.

7.1.1 Ambient Particulate Matter Concentration as a Surrogate for Particulate Matter Dosage

The health effects of PM experienced by an individual depend upon the mass, size and composition of those particles deposited within various regions of the respiratory tract during the time interval of interest. The amount of this potential dose will depend on the concentration inhaled (e.g., the instantaneous personal exposure); the ventilation rate (a function of physical activity and basal metabolism); and the fractional deposition, which is a function of ventilation rate, mode of breathing (e.g., oral or nasal), and any alterations due to lung dysfunction. If all people had identical ventilation rates and deposition patterns, then the potential-dosage distribution could be linearly scaled to the personal exposure distribution which would serve as a suitable primary surrogate. The usage of ambient PM concentration in health studies as a surrogate for personal PM exposure, and thereby a secondary surrogate for the PM dosage, would be suitable if ambient concentration was also linearly related to the personal exposure (Mage, 1983).

Adult ventilation rates are lowest (mean ≈ 6 L/min) during the night while asleep, at a maximum (mean ≈ 12 L/min; peak ≈ 60 L/min) during the day while awake (Adams, 1993), and in phase with PM exposure, which is also lower at night than during the day (Clayton et al., 1993). Consequently, the product of the 24-h average PM exposure, the 24-h average ventilation rate, and the average deposition parameter for the average ventilation would seriously under-predict the amount of PM deposited in the respiratory tract (Mage, 1980).

In practice, when relating human health to PM pollution variables (as in Chapter 12) one is forced to use time-weighted-average (TWA) ambient PM concentration as a surrogate for PM exposure and PM dosage because only fragmentary data are typically available on personal exposures to PM in populations. Data are also limited on ventilation rates as a function of basal

metabolism and physical activities (Adams, 1993), as are data on pulmonary deposition rates of particles people are inhaling, since the size distribution is unknown and deposition is affected by unmeasured individual physiological parameters. According to Hodges and Moore (1977), "even when an explanatory variable (ambient PM concentration) can be measured with negligible error it may often be standing as a proxy for some other variable (dosage) which cannot be measured directly, and so it (dosage) is subject to measurement error". Pickles (1982) shows "that (such) uncertainties in air pollution levels lead to two kinds of error in the air pollution/mortality regression coefficient - a systematic underestimate and a random scatter". In addition, measurement error can also bias a threshold in the dose-response function towards zero (Yoshimura, 1990).

In the sections that follow, the relationships between ambient PM concentration, indoor PM concentrations and personal exposures to PM are discussed in detail. The following five caveats should be kept in mind while reading this chapter:

1. Ambient PM concentrations are often measured as a 24-h time-weighted-average (TWA) expressed as $\mu g/m^3$. This quantity, by necessity, is assumed to be a surrogate for the mass of ambient PM deposited in people's respiratory tracts per unit body weight, expressed as µg/kg-day.

2. This daily quantity of ambient PM deposited per unit body weight is in turn a surrogate for the amount of the true (but unknown) species and/or size fraction of the total PM that is the specific etiologic toxic agent(s) that act by a presently unknown mechanism. This latter quantity should be the independent variable for delineating underlying relationships between ambient PM TWA concentrations to the health indices used as the dependent variables.

3. Virtually all analyses and discussions of exposure presented here are based on personal exposure to PM of non-smokers. Only Dockery and Spengler (1981b) included 6 smokers out of 37 subjects. Smokers are often excluded from these studies because a personal exposure monitor (PEM) on a smoker will not capture the main-stream tobacco smoke that is directly inhaled. In Section 7.2 on indoor air pollution, it is shown that side-stream environmental tobacco smoke (ETS) is the largest identifiable indoor source of PM where smoking occurs. For the average smoker, the amount of direct inhalation (several milligrams of PM per cigarette) can be two-to-three orders of magnitude greater than the microgram amounts of ETS which the PEM captures (Federal Trade Commission, 1994). The relationships presented below, of ambient PM concentration to individual total PM exposure, therefore only apply to non-smokers.

4. A total TWA personal exposure to PM (ambient PM plus indoor PM) will be a poor surrogate for the personal exposure to PM of ambient origin for those people whose

personal exposures are dominated by indoor (residential and occupational) sources, such as ETS.

5. All studies of indoor concentrations and personal exposures described below evaluated subjects recruited either in a nonrandom manner or in a scientific probability sampling scheme. In the former case, the results cannot be extrapolated with confidence beyond the subjects themselves. In the latter case, the results can be extrapolated with a known confidence to the target population from which the sample was drawn. However, in both cases, there is a cohort of people who are nonresponders. If the reason for their refusal to participate in the survey is directly or indirectly related to their PM exposure, then the study results represent a sample with a bias of unknown sign and magnitude.

7.1.2 General Concepts for Understanding Particulate Matter Exposure and Microenvironments

Particulate matter represents a generic class of pollutants which requires a different interpretation of exposure in contrast to that for the other specific criteria gaseous pollutants, such as CO (Mage, 1985). Whereas a molecule of CO emitted from a motor vehicle is indistinguishable from a molecule of CO emitted from a fireplace, a 1-µm aerodynamic diameter (AD) particle emitted from a motor vehicle and a 1-µm particle emitted from a fireplace can have a different shape, mass, chemical composition, and/or toxicity. Thus, a "particle" can be a single entity, or an agglomeration of smaller particles, such as a small Pb particle bound to a larger crustal particle. Furthermore, indoor sources of particles produce a wide variety of particles of varying size and composition that people are exposed to, as shown in Figure 7-1 (Owen et al., 1992). Given that the health effects of inhalation of any particle can depend upon its mass and chemical composition, it would be of use to measure PM exposure in terms of mass and chemical composition as a function of size distribution (Mage, 1985).

The total PM exposure of an individual during a period of time is composed of exposure to many different particles from various sources in different microenvironments (µE). A μE was defined by Duan (1982) as "a chunk of air space with homogeneous

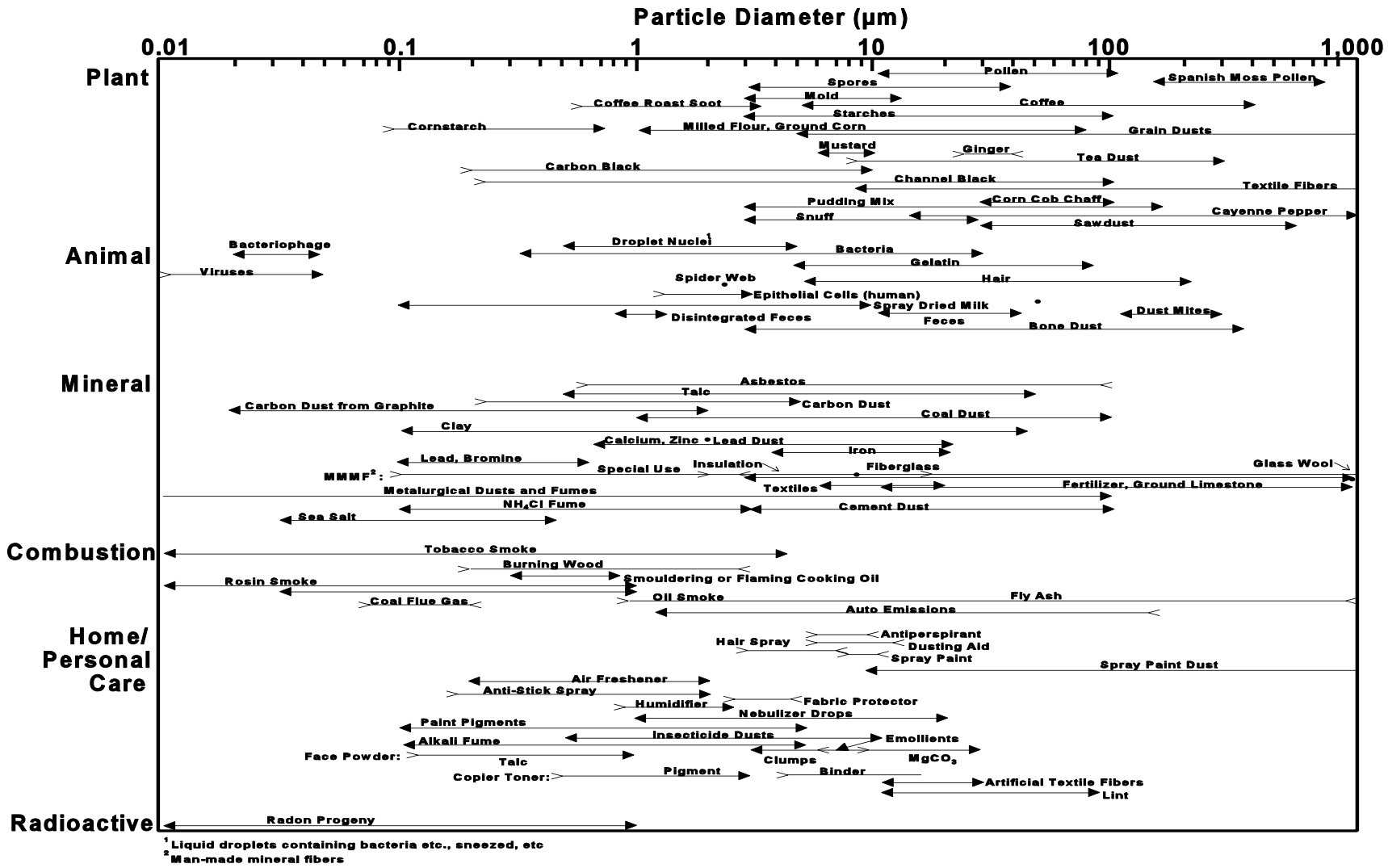


Figure 7-1. Sizes of various types of indoor particles.

Source: Owen et al. (1992).

pollutant concentration"; it has also been defined (Mage, 1985) as a volume in space, during a specific time interval, during which the variance of concentration within the volume is significantly less than the variance between that µE and its surrounding µEs. For example, a kitchen with a wood stove can constitute a single µE for total PM when the stove is off, and all people in the kitchen would have similar PM exposures. When the stove is in operation, the kitchen could have a significant vertical PM concentration gradient and a child on the floor in a far corner and an adult standing at the stove could be exposed to significantly different PM concentrations.

In a given µE, such as one in the kitchen example, the particles may come from a wide variety of sources. PM may be generated from within (e.g. the stove, deep frying, burning toast), from without (ambient PM entering through an open window), from another indoor µE (cigarette smoke from the living room), or from a personal activity that generates a heterogeneous mix of PM (sweeping the kitchen floor and resuspending a mixture of PM from indoor and outdoor sources that had settled out).

In general, as people move through space and time, they pass through a series of µEs and their average total exposure (X µg/m^3) to PM for the day can be expressed by the following equation,

$$X = \Sigma X_i t_i / \Sigma t_i \qquad (7\text{-}1)$$

where X_i is the total exposure to PM in the i^{th} µE, visited in sequence by the person for a time interval t_i (Mage, 1985).

With appropriate averaging over sets of 4 classes of µEs (e.g., indoors, ambient-outdoors, occupational, and in-traffic) Equation 7-1 can be simplified as follows (Mage, 1985):

$$X = (X_{in} t_{in} + X_{out} t_{out} + X_{occ} t_{occ} + X_{tra} t_{tra}) / T \qquad (7\text{-}2)$$

where each value of X is the mean value of total PM concentration in the µE class *while the subject is in it*, time (t) is the total time the subject is in that µE during the day, and T is equal to the sum of all times (usually one day). Similar equations may be written for personal exposures to particles from specific sources (e.g., diesel soot), for specific chemicals (e.g., Pb), or for specific size intervals (PM $\leq$ 2.5 µm AD).

Many excellent studies have reported data on air quality concentrations in µE settings that do not meet a rigorous definition of an exposure, which requires actual occupancy by a person

(Ott, 1982). Section 7.2, on Indoor Concentrations and Sources of PM, cites Thatcher and Layton (1995) who report that "merely walking into a room increased the particle concentration by 100%". Consequently, an integrated measurement of air quality in an enclosed space that includes time when it is unoccupied may not be a valid measure that can be used to estimate an exposure while occupied. If this measure includes periods of time when the space is unoccupied, it will tend to be biased low as a measure of the exposure within it during periods of occupancy. For example, it is incorrect to associate an average PM exposure to a person while cooking at a stove in a kitchen with a kitchen concentration measurement that is influenced by periods when the stove was off (Smith et al., 1994).

The literature on 24-h average PM concentrations in indoor μEs, such as residential settings, is treated separately in Section 7.2, as is done for 24-h average ambient PM concentrations in Chapter 6. In the exposure portion of this chapter, specific reference is made to some studies where simultaneous personal PM exposures and indoor PM measurements have been made, so that the relationship between indoor concentration and personal exposure can be examined.

In practice, a cascade sampler can collect ambient PM samples by size fractionation for separate chemical analyses, but such a complete definition of personal exposure to PM by chemistry and size is difficult to obtain. Although some personal monitors can be equipped with a cyclone or impactor separator and several filters to capture several PM sizes (e.g., <2.0 μm, 2.0 to 10 μm, and >10 μm; Tamura et al., 1996), most published studies of PM exposure used a PEM with a single integrated measurement of particle mass collected (e.g., <2.5 μm or <10 μm). Consequently, health studies on individuals are usually only able to develop associations between their observed health effects and their observed exposure expressed as an integral mass of PM collected and its average chemical composition.

Health studies on populations can make multiple measurements of ambient and indoor PM concentrations simultaneously (e.g., $PM_{2.5}$, PM_{10}, TSP) along with components of PM, such as polycyclic aromatic hydrocarbons (PAHs), to help understand the size distribution and chemistry of the particles in the ambient and indoor atmospheres.

7.1.3 Summary of State-of-Knowledge in the 1982 Criteria Document

In 1982 it was known, from personal monitoring and indoor monitoring, that SO_2 is almost always lower indoors than outdoors because of the virtual absence of indoor sources and the presence of sinks for SO_2 in indoor settings (exceptions can occur if high sulfur coal or kerosene are used as fuel in a poorly vented stove or space heater). However, this relationship does not hold for PM, as the indoor and personal monitoring data show both higher- and lower-than ambient PM concentrations in indoor settings as a function of particle size and human activity patterns.

The largest coarse mode particles (>10 μm), which are generally of nonanthropogenic origin (wind blown dust, etc.), require turbulence to provide vertical velocity components greater than their settling velocity to allow them to remain suspended in the air (Figure 7-1). Outdoor particles enter into an indoor setting either by bulk flow, as through an open window, in which all particles can enter at the inlet condition, or by pressure driven drafts and diffusional flows through cracks and fissures in the barriers of the building envelope when all windows are closed. In the latter mode of entry, velocities are relatively lower, thereby settling out the largest coarse particles (>25 μm AD) in the passage through the barriers (Thatcher and Layton, 1995).

Indoor settings are usually quiescent (Matthews et al., 1989), and ambient particles that enter indoors quickly settle out by gravity or electrostatic forces, leading to familiar dust layers on horizontal surfaces and vertical TV screens that require constant cleaning (Raunemaa et al., 1989). However, human activity in indoor settings, such as smoking, dusting, vacuuming and cooking, does generate fine particles (<2.5 μm) and coarser particles (>2.5 μm) and resuspends coarse particles (>10 μm) that previously had settled out (Thatcher and Layton, 1995; Litzistorf et al., 1985).

Only three studies of personal PM exposures, compared to ambient PM concentrations, were referenced in the 1982 Criteria Document (U.S. Environmental Protection Agency, 1982). Binder et al. (1976) reported that "outdoor air measurements do not accurately reflect the air pollution load experienced by individuals who live in the area of sampling", in a study in Ansonia, CT, where personal exposures to PM_5 were double the outdoor PM concentrations measured as TSP (115 versus 58 $\mu g/m^3$). Spengler et al. (1980) was cited as reporting that "there was no correlation [$R^2 = 0.04$] between the outdoor level [of respirable particles] and the personal exposure of individuals" in a study in Topeka, KS. Figure 7-2, from Repace et al.

(1980), was cited as an example of the variability of PM exposures which show very little influence of ambient concentration. Thus, at the time of the 1982 Criteria Document, two major factors were known to influence ambient PM relationships to indoor PM air quality: (1) the variability of indoor levels of PM compared to outdoor concentrations as a function of particle size (e.g., fine indoor ≥ fine outdoor, and coarse indoor < coarse outdoor); and (2) the variation of exposures of individuals related to different activities involved in local generation of particles in their immediate surroundings (smoking, traffic, dusting and vacuuming at home, etc.). This understanding was summarized on pg. 5-136 of the 1982 Criteria Document, as follows:

- long term personal exposures to fine fraction PM (<2.5 μm) of outdoor origin, may be estimated by ambient measurements of the <2.5 μm PM fraction.
- Personal activities and indoor concentrations cause personal exposures to PM to vary substantially. Ambient measurements appear to be a poor predictor of personal exposure to PM.
- Tobacco smoke is an important contributor to indoor concentrations and personal exposures where smoking takes place (U.S. Environmental Protection Agency, 1982).

7.2 INDOOR CONCENTRATIONS AND SOURCES OF PARTICULATE MATTER

7.2.1 Introduction

Although EPA regulates particles in ambient air, which excludes the air internal to buildings, it is still important to consider indoor air. Most people spend most of their time indoors. A U.S. Environmental Protection Agency (1989) report indicates that U.S. residents spend 85.2% of their time indoors, 7.4% in or near a vehicle, and only 7.4% outdoors. Also, it is important to understand how outdoor particles are affected as they cross building envelopes. For a home with no indoor sources, how much protection is offered against particles of various size ranges? How do parameters such as volume of the house, air exchange rate, cleaning frequency and methods, and materials in the home affect

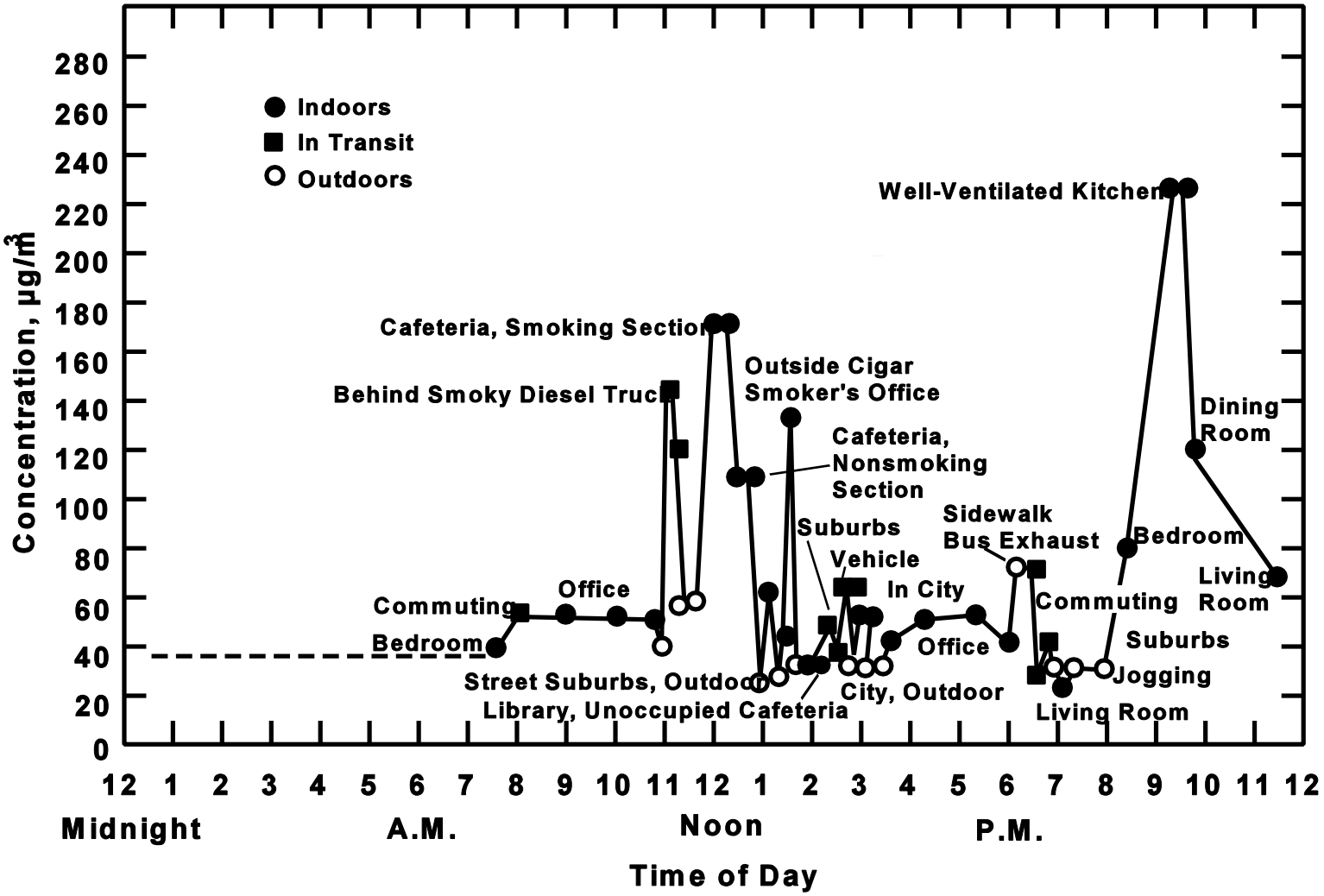


Figure 7-2. An example of personal exposure to respirable particles.

Source: Repace et al. (1980).

concentrations of particles of outdoor origin? This section has several parts that address these questions.

The first part (7.2.2; 7.2.3; and 7.2.4) deals with field studies of particles indoors and outdoors, focussing mainly on large-scale surveys of many homes and buildings. Besides presenting observed indoor and outdoor particle concentrations, information on important parameters such as air exchange rates, source emission rates, and deposition rates is also reported. This section also discusses a few studies dealing with inorganic and organic constituents of particles, as well as other considerations such as the role of house dust in exposure to metals. Section 7.2.3 provides a brief introduction to indoor air quality models. Finally, Section 7.2.4 summarizes the main findings.

The second part (7.2.5) is a discussion of bioaerosols from plants, molds, insects, etc. Although these sources of PM are uncontrolled by EPA, they affect measured PM indoors and can potentiate the effects of PM from other sources through allergenic properties.

In keeping with EPA's regulatory responsibilities, the many studies in industrial workplaces and the "dusty trades" are omitted, as are studies of lead (Pb) in indoor locations, since lead is a separate criteria pollutant and such studies are reviewed in a separate lead criteria document (U.S. Environmental Protection Agency, 1986b).

7.2.2 Concentrations of Particles in Homes and Buildings

At least seven major reviews of field studies of indoor particles have been published since 1980 (Sterling et al., 1982; National Research Council, 1986; Repace, 1987; Guerin et al., 1992; U.S. Environmental Protection Agency, 1992; Holcomb, 1993; Wallace, 1996). The last of these reviews reports on several recently completed important studies, including EPA's major probability-based PTEAM Study. Since the two microenvironments where people spend the most time are (a) home and (b) work or school, studies of these environments are summarized in turn, with emphasis on the former.

7.2.2.1 Particle Concentrations in Homes: Large-Scale Studies in the United States

There have been three large-scale studies (greater than 150 homes) of airborne particles inside U.S. homes. In chronological order, these are:

1. The Harvard Six-City study, carried out by the Harvard School of Public Health from 1979 through 1988, with measurements taken in 1,273 homes;

2. The New York State ERDA study, carried out by Research Triangle Institute (RTI) in 433 homes in two New York State counties during 1986;

3. The EPA Particle TEAM (PTEAM) study, carried out by RTI and Harvard School of Public Health in 178 homes in Riverside, CA in 1990.

The findings of each are discussed in detail, since these studies present the most complete investigations to date of indoor and outdoor concentrations of particles.

7.2.2.1.1 The Harvard Six-City Study

The Harvard Six-City Study is a prospective epidemiological study of health effects of particles and sulfur oxides. Focused mainly on children, it has included pulmonary function measurements on more than 20,000 persons in the six cities, chosen to represent low (Portage, WI and Topeka, KS), medium (Watertown, MA and Kingston-Harriman, TN), and high (St. Louis, MO and Steubenville, OH) outdoor particle and sulfate concentrations.

The study took place in two measurement phases. The first involved monitoring of about 10 homes in each city for respirable particles ($PM_{3.5}$), with measurements made every sixth day (24-h samples) for one to two years. In the second phase, a larger sample of 200 to 300 homes was selected from each city, with week-long $PM_{2.5}$ samples collected both indoors and outdoors during two weeks of sampling in summer and winter. Ultimately, more than 1,200 homes were monitored in this way.

Spengler et al. (1981) described the first five years of the study. During the Phase I period, pulmonary function measurements were made for 9,000 adults, and 11,000 children in grades 1 through 6. In each home, a 24-h sample (beginning at midnight) was collected every sixth day, using a cyclone sampler with a cut point of ≈3.5 μm at a flow rate of 1.7 Lpm. About 10 sites in each city were kept in operation for two years. The annual mean indoor and outdoor $PM_{3.5}$ concentrations are shown in Figure 7-3. The indoor concentrations exceeded the outdoor levels in all cities except Steubenville, OH, where the outdoor levels of about 46 $\mu g/m^3$ slightly exceeded the indoor mean of about 43 $\mu g/m^3$. The authors noted that the major source of indoor particles was cigarette smoke, and categorized their data by number of smokers in the home (Table 7-1).

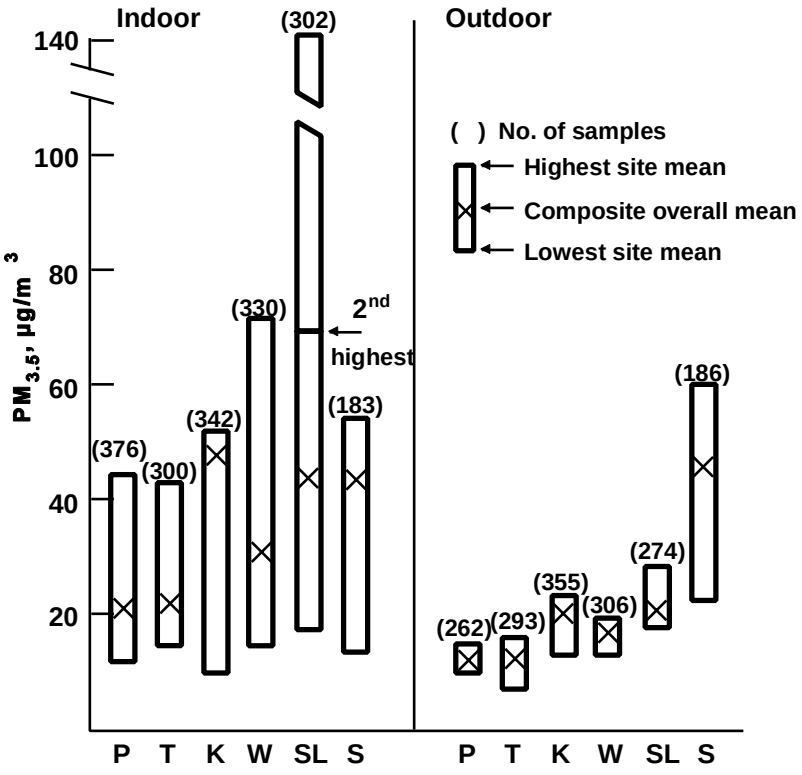


Figure 7-3. The annual mean concentration of respirable particles ($PM_{3.5}$) for the highest and lowest site from the network of indoor and outdoor monitors in each city (P-Portage, T-Topeka, K-Kingston/Harriman, W-Watertown, SL-St. Louis, S-Steubenville) in the Harvard Six-City study. Overall composite mean and the number of samples are also shown.

Source: Spengler et al. (1981).

TABLE 7-1. CONCENTRATIONS OF PARTICLES ($PM_{3.5}$) IN HOMES OF CHILDREN PARTICIPATING IN THE HARVARD SIX-CITY STUDY

Location	No. of Homes	No. of Samples	Mean (SD) ($\mu g/m^3$)
Indoors			
No smokers	35	1,186	24.4 (11.6)
One smoker	15	494	36.5 (14.5)
Two or more smokers	5	153	70.4 (42.9)
Outdoors	55	1,676	21.1 (11.9)

Source: Spengler et al. (1981).

Dockery and Spengler (1981a) provided additional data analyses drawn from the same study but including data from 68 homes compared to the 55 reported on in Spengler et al. (1981). Annual (every sixth day) mean indoor $PM_{3.5}$ concentrations (in $\mu g/m^3$) were 20 and 23 in the two "clean" locations (Portage and Topeka); 31 and 36 in the two "medium" locations (Watertown and Kingston-Harriman); and 39 and 47 in the two "dirty" locations (Steubenville and St. Louis). Outdoor $PM_{2.5}$ concentrations measured by dichotomous samplers every other day ranged from 13 $\mu g/m^3$ in Portage and Topeka to 20 $\mu g/m^3$ in St. Louis, 24 $\mu g/m^3$ in Kingston-Harriman, and 36 $\mu g/m^3$ in Steubenville (Spengler and Thurston, 1983). A mass balance model allowed estimation of the impact of cigarette smoking on indoor particles. Long-term mean infiltration of outdoor $PM_{3.5}$ was estimated to be 70% for homes without air conditioners, but only 30% for homes with air conditioners. A contribution of 0.88 $\mu g/m^3$ per cigarette (24-h average) was estimated for homes without air conditioning; for homes with air conditioning, it increased to 1.23 $\mu g/m^3$ per cigarette. A residual amount of 15 $\mu g/m^3$ not explained by the model was attributed to indoor sources such as cooking, vacuuming and dusting.

From the one to two years of indoor-outdoor data on 57 homes in the six cities, Letz et al. (1984) developed an equation relating indoor to outdoor particle concentrations:

$$C_{in} = 0.385\ C_{out} + 29.4\ (\text{Smoking}) + 13.8.$$

Thus, homes with smokers had a $PM_{3.5}$ ETS component of 29.4 $\mu g/m^3$. The residual of 13.8 $\mu g/m^3$ was assumed to be due to other household activities.

Neas et al. (1994) presented summary results for the entire Phase 2 of the Six-City Study (1983 to 1988). In Phase 2, for 1,237 homes containing white, never-smoking children, 7 to 11 years old at enrollment, three questionnaires were completed and two weeks of summer and winter monitoring indoors and outdoors for $PM_{2.5}$ was done, using the Harvard $PM_{2.5}$ impactor. At the start of the indoor monitoring study, 55% of the children were exposed to ETS in the home, and 32% were exposed to two or more smokers. Household smoking status changed for 173 children, (13% of smoking households ceased to smoke, and 15% of the nonsmoking households became smoking ones). The annual (winter and summer) household $PM_{2.5}$ mean concentration for the 580 children living in consistently smoking households was 48.5 ± 1.4 (SE) $\mu g/m^3$ compared to 17.3 ± 0.5 $\mu g/m^3$ for the 470 children in consistently nonsmoking

households. Among the 614 exposed children for whom complete information on smoking consumption was available, 36% were exposed to < 1/2 pack daily, 40% to 1/2 to 1 pack daily, and 25% to >1 pack daily. The distribution of household concentrations for children in these smoking categories is shown in Figure 7-4.

Spengler et al. (1985) reported on the Kingston-Harriman, TN data from the Six-City Study. Of 101 participants, 28 had cigarette smoke exposure at home, and each had an indoor and personal monitor (cutpoints of 3.5 μm). Each town had a centrally located outdoor dichotomous sampler providing two size fractions (2.5 μm and 15 μm). Both towns had similar outdoor $PM_{2.5}$ concentrations of 18 $\mu g/m^3$, so the values were pooled for subsequent analyses. Indoor concentrations averaged 42 ± 2.6 (SE) $\mu g/m^3$. Indoor values in homes with smoking averaged 74 ± 6.6 $\mu g/m^3$, compared to 28 ± 1.1 $\mu g/m^3$ in homes without smoking ($p < 0.0001$). No significant correlations between indoor and outdoor concentrations were observed.

Lebret et al. (1987) reported on the Watertown, MA portion of the Six-City Study where 265 homes were monitored for two one-week periods. Homes with smoking averaged 54 $\mu g/m^3$ (N = 147 and 152 during weeks 1 and 2), while homes without smoking averaged 21.6 $\mu g/m^3$ (N = 70 and 74). The effect of smoking one cigarette/day was estimated at 0.8 $\mu g/m^3$ of $PM_{2.5}$.

Spengler et al. (1987) reported on a new round of measurements in three Six-City Study communities: Watertown, MA; St. Louis, MO; and Kingston-Harriman, TN. In each community, about 300 children were selected to take part in a year-long diary and indoor air quality study. $PM_{2.5}$ measurements were taken indoors at home for two consecutive weeks in winter and in summer, using the automated Harvard sampler which collected an integrated sample for the week except for 8 a.m. to 4 p.m. weekday periods when the child was at school. During this 40-h period, samples were taken in one classroom in each of the elementary schools involved. Results were presented for smoking and non-smoking homes in each city by season (Figure 7-5); the authors noted that mean concentrations in homes with smokers were about 30 $\mu g/m^3$ greater than homes without smokers, the difference being greater in winter than in summer for all cities.

Santanam et al. (1990) reported on a more recent and larger-scale monitoring effort in Steubenville and Portage as part of the Six-City Study; 140 homes in each city, equally

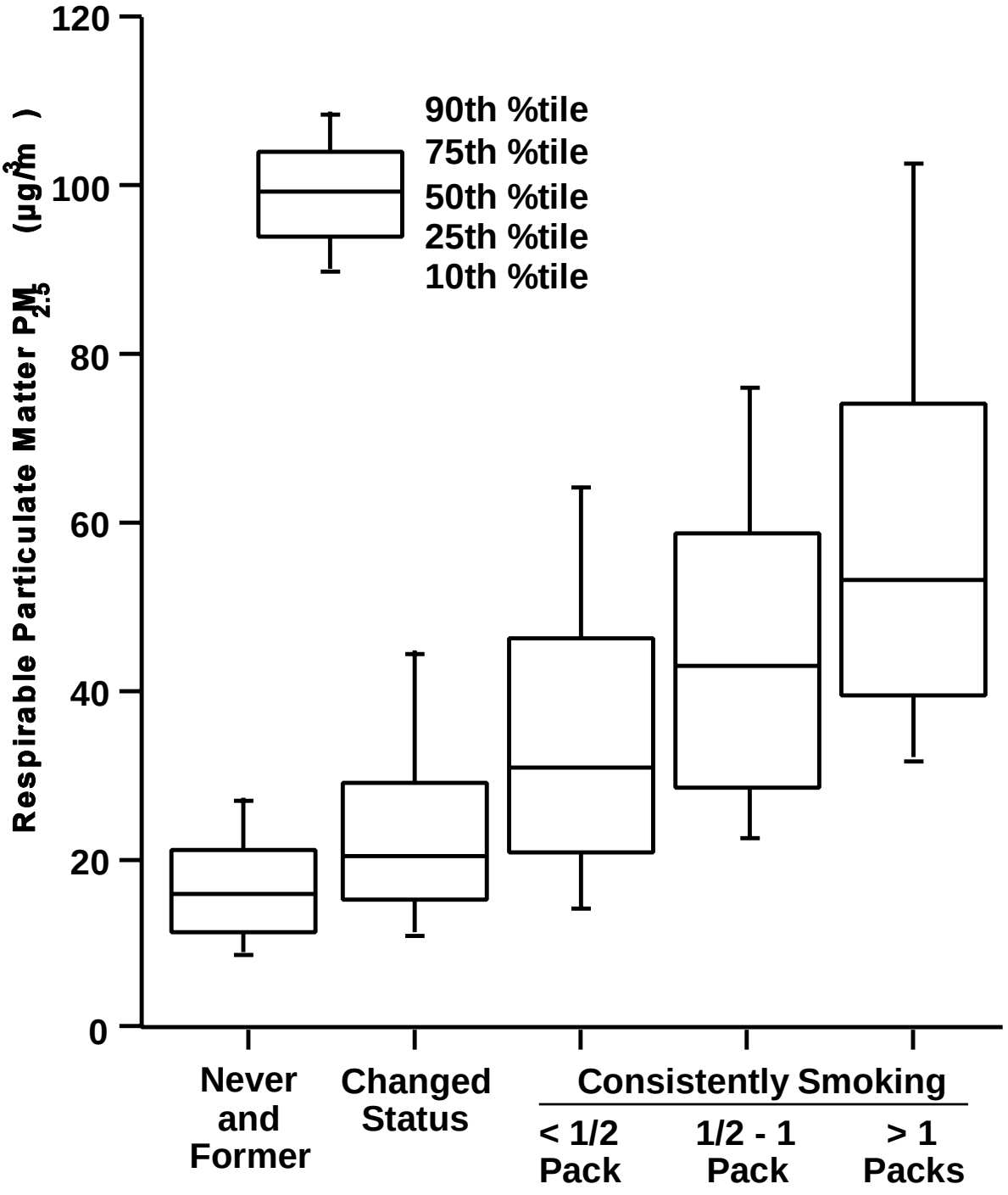


Figure 7-4. Distribution percentiles for annual average concentrations of indoor respirable particulate matter ($PM_{2.5}$) by household smoking status and estimated number of cigarette packs smoked in the home during Phase 2 Harvard Six-City study.

Source: Neas et al. (1994).

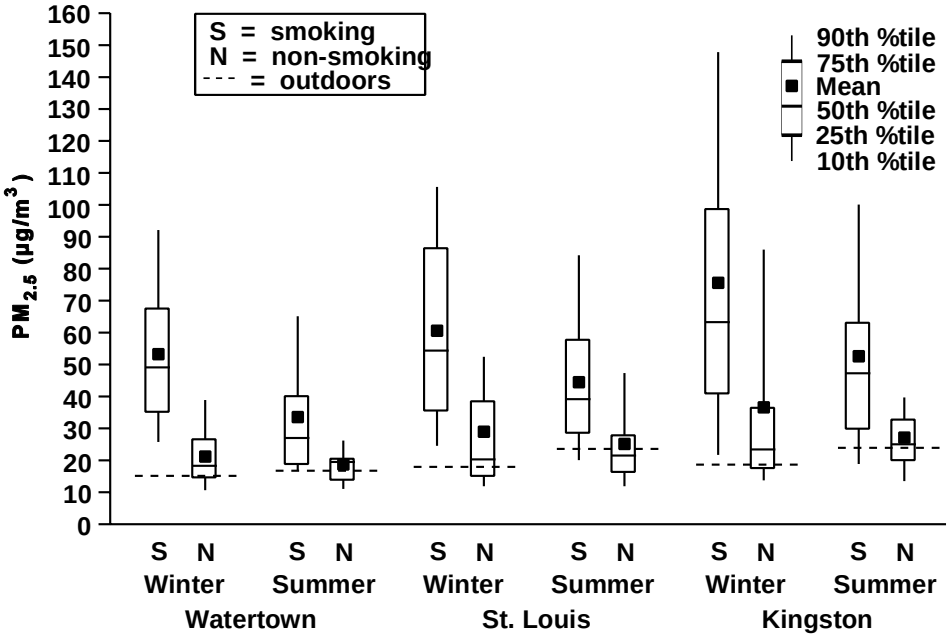


Figure 7-5. $PM_{2.5}$ ($\mu g/m^3$) in smoking (S) and nonsmoking (N) homes in three of the Harvard Six-City Study sites.

Source: Spengler et al. (1987).

distributed among households with and without smoking were monitored for one week in summer and in winter. The Harvard impactor sampler was used to collect $PM_{2.5}$ samples between 4 p.m. and 8 a.m. on weekdays and all day on weekends, corresponding to likely times of occupancy for school-age children. Outdoor samples were collected from one site in each city. Target elements were determined by XRF. A source apportionment using principal components analysis (PCA) and linear regressions on the elemental data were carried out (Table 7-2a,b). Cigarette smoking was the single largest source in smokers' homes, accounting for 20 to 27 µg/m^3 indoor $PM_{2.5}$ in Steubenville (Table 7-2a) and 10 to 25 µg/m^3 in Portage (Table 7-2b). Wood smoke was estimated to account for about 4 µg/m^3 indoors and outdoors in Steubenville in winter, but only for about 1 µg/m^3 indoors and outdoors in Portage. Sulfur-related sources accounted for 8 to 9 µg/m^3 indoors and 16 µg/m^3 outdoors in Steubenville in the summer, but were apparently not important in winter. Auto-related sources accounted for 2 to 5 µg/m^3 in the two cities. Soil sources

TABLE 7-2a. RECONSTRUCTED SOURCE CONTRIBUTIONS TO INDOOR $PM_{2.5}$ MASS FOR STEUBENVILLE, OH[1]

	WINTER			SUMMER		
Source	Smokers' Homes	Non-Smokers' Homes	Outdoor Site	Smokers' Homes	Non-Smokers' Homes	Outdoor Site
Soil	7.9 (3.45)	17.6 (3.45)	9.6 (1.79)	NS	NS	NS
Wood smoke	9.5 (4.15)	21.2 (4.15)	23.0 (4.31)	NS	NS	NS
O.C.-I	10.3 (4.47)	22.9 (4.47)	24.8 (4.65)	NS	NS	NS
Tobacco Smoke	45.6 (19.9)	NA	NA	53.7 (26.8)	NA	NA
Sulfur-related	NS	NS	NS	17.8 (8.90)	33.3 (8.23)	52.5 (15.5)
Auto-related	NS	NS	NS	7.3 (3.65)	14.8 (3.65)	5.3 (1.55)
O.C.-II	NS	NS	NS	8.8 (4.40)	16.5 (4.07)	26.0 (7.67)
Indoor dust	NS	NS	NA	7.4 (3.70)	15.0 (3.70)	NA
Unexplained	26.7 (11.6)	38.3 (7.47)	42.6 (7.95)	5.0 (2.4)	20.4 (5.05)	16.2 (4.78)
Total	100 (43.57)	100 (19.54)	100 (18.7)	100 (49.85)	100 (24.7)	100 (29.5)

[1]All entries in % ($\mu g/m^3$)
NS = not significant.
NA = not applicable.
O.C.-I: Iron and steel, and auto-related sources.
O.C.-II: Iron and steel, and soil sources.

Source: Santanam et al. (1990).

TABLE 7-2b. RECONSTRUCTED SOURCE CONTRIBUTIONS TO INDOOR $PM_{2.5}$ MASS FOR PORTAGE, WI[1]

	WINTER			SUMMER		
Source	Smokers' Homes	Non-Smokers' Homes	Outdoor Site	Smokers' Homes	Non-Smokers' Homes	Outdoor Site
Sulfur-related	13.2 (4.56)	30.7 (4.56)	39.2 (4.04)	23.3 (5.80)	38.1 (5.30)	45.8 (6.23)
Auto-related	5.1 (1.78)	12.0 (1.78)	17.3 (1.78)	18.1 (4.50)	29.6 (4.12)	35.6 (4.84)
Soil	3.8 (1.31)	8.8 (1.31)	13.4 (1.38)	7.5 (1.86)	13.4 (1.86)	16.5 (2.25)
Tobacco Smoke	71.0 (24.6)	NA	NA	40.1 (9.99)	NA	NA
Wood smoke	2.7 (0.94)	6.3 (0.94)	13.0 (1.34)	NA	NA	NA
Unexplained	4.2 (1.38)	42.2 (6.23)	17.1 (1.80)	11.0 (2.75)	18.9 (2.62)	2.10 (0.28)
Total	100 (34.6)	100 (14.8)	100 (10.3)	100 (24.9)	100 (13.9)	100 (13.6)

[1]All entries in % ($\mu g/m^3$)
NA = not applicable.

Source: Santanam et al. (1990).

accounted for only about 1 to 3 μg/m^3 of indoor and outdoor $PM_{2.5}$ concentrations. Nonsmoking homes in both cities had indoor mean $PM_{2.5}$ concentrations very close to the outdoor mean concentrations. Quite large percentages of particle concentrations were due to unexplained sources.

7.2.2.1.2 The New York State ERDA Study

Sheldon et al. (1989) studied $PM_{2.5}$ and other pollutants in 433 homes in two New York State counties. One goal of the study was to determine the effect of kerosene heaters, gas stoves, wood stoves or fireplaces, and cigarette smoking on indoor concentrations of combustion products. A stratified design included all 16 combinations of the four combustion sources and required about 22,000 telephone calls to fill all cells. The sampler was a portable dual-nozzle impactor developed at Harvard University. Two oiled impactor plates in series were used to reduce the probability that some particles larger than 2.5 μm would reach the filter. Samples were collected in the main living area and in one other room (containing a combustion source if possible) using a solenoid switch to collect alternate 15-min samples over a 7-day period. Outdoor samples were collected at a subset of 57 homes. All samples were collected during the winter (January to April) of 1986.

$PM_{2.5}$ mean concentrations indoors for all homes, with and without any combustion sources, were approximately double those outdoors in both counties (Table 7-3). However, in homes without combustion sources, $PM_{2.5}$ concentrations were approximately equal (Leaderer et al., 1990). Of the four combustion sources, only smoking created significantly higher indoor $PM_{2.5}$ concentrations in both counties (Table 7-4). Use of kerosene heaters was associated with significantly higher concentrations in Suffolk (N = 22) but not in Onondaga (N = 13). Use of wood stoves/fireplaces and gas stoves did not significantly elevate indoor concentrations in either county.

Leaderer et al. (1990) extended the analysis of these data by collapsing the gas stove category, reducing the number of categories from 16 to 8 (Table 7-5). By inspection of Table 7-5, it is clear that smoking was the single strongest source of indoor fine particles, with geometric means of indoor PM ranging from 28.5 to 61.4 μg/m^3, whereas the four nonsmoking categories ranged from 14.1 to 22.0 μg/m^3.

TABLE 7-3. WEIGHTED SUMMARY STATISTICS BY NEW YORK COUNTY FOR RESPIRABLE SUSPENDED PARTICULATE ($PM_{2.5}$) CONCENTRATIONS ($\mu g/m^3$)

	Main Living Area		Outdoors	
	Onondaga	Suffolk	Onondaga	Suffolk
Percent Detected	98.9	99.6	100	100
Sample Size	224	209	37	20
Population Estimate	94,654	286,580		
Arithmetic Mean ($\mu g/m^3$)	36.7[a]	46.4	16.8	21.8
Arithmetic Standard Error ($\mu g/m^3$)	2.14	2.77	1.00	4.54
Geometric Mean ($\mu g/m^3$)	25.7[a]	35.9	15.8	18.6
Geometric Standard Error	1.07	1.06	1.06	1.11
Minimum ($\mu g/m^3$)	0.72	2.18	6.32	12.0
Maximum ($\mu g/m^3$)	172	284	28.4	106
Percentiles				
10th	9.93	13.8		
16th	11.2	16.8		
25th	13.5	18.9	12.8	13.6
50th (median)	23.9	33.6	15.1	16.7
75th	48.4	62.8	20.5	22.3
84th	68.0	76.6		
90th	85.2	89.4		
95th	112	112		
99th	136	155		

[a] Significantly different between counties at 0.05 level.

Source: Sheldon et al. (1989).

Leaderer and Hammond (1991) continued analysis of the New York State data by selecting a subset of 96 homes for which both nicotine and $PM_{2.5}$ data were obtained. In the 47 homes where nicotine was detected (detection limit = 0.1 $\mu g/m^3$), the mean concentration of RSP was 44.1 (± 25.9 SD) $\mu g/m^3$ compared to 15.2 (± 7.4) $\mu g/m^3$ in the 49 homes without detected nicotine. Thus, homes with smoking had an increased weekly mean $PM_{2.5}$ concentration of about 29 $\mu g/m^3$. Imperfect agreement with reported smoking was observed, with nicotine being measured in 13% of the residences that reported no smoking, while nicotine was not detected in 28% of the residences that reported smoking. A regression on

TABLE 7-4. WEIGHTED ANALYSIS OF VARIANCE OF RESPIRABLE SUSPENDED PARTICULATE ($PM_{2.5}$) CONCENTRATIONS ($\mu g/m^3$) IN THE MAIN LIVING AREA OF HOMES VERSUS SOURCE CLASSIFICATION

	F Value	Probability	Coefficient
Onondaga ($R^2 = 0.17$)			
Model	20.5	0.00	
Independent variables:			
Intercept			20.3
Gas stove	1.87	0.17	5.25
Kerosene heater	1.06	0.30	5.05
Tobacco smoking	81.6	0.00	45.1
Wood stove/fireplace	2.42	0.12	7.81
Suffolk ($R^2 = 0.21$)			
Model	36.9	0.00	
Independent variables:			
Intercept			26.1
Gas stove	0.13	0.72	−1.52
Kerosene heater	12.0	0.00	30.1
Tobacco smoking	114	0.00	46.8
Wood stove/fireplace	0.71	0.40	9.88

Source: Sheldon et al. (1989).

TABLE 7-5. RESPIRABLE SUSPENDED PARTICULATE ($PM_{2.5}$) CONCENTRATION ($\mu g/m^3$) IN HOMES BY SOURCE CATEGORY

	Suffolk			Onondaga		
Source	N	Mean	Standard	N	Mean	Standard
None	30	17.3	1.7	45	14.1	1.7
W	15	18.1	1.6	16	19.1	1.7
K	7	22.0	1.6	4	21.2	1.0
S	61	49.3	1.8	80	36.5	2.4
KW				4	19.7	1.5
SW	29	38.0	1.8	31	33.9	2.2
SK	23	61.4	2.0	4	35.3	1.5
SKW	6	30.3	1.4	4	28.5	1.6
Outdoor	19	16.9	1.3	36	15.8	1.5

Abbreviations: W = woodstove; K = kerosene heater; S = tobacco smoking.

Source: Leaderer et al. (1990).

all (smoking and nonsmoking) homes of $PM_{2.5}$ on total number of cigarettes smoked during the week (T) gave the result:

$$PM_{2.5} = 17.7 + 0.322T \text{ (N = 96; } R^2 = 0.55).$$

For the subset of 47 homes with measured nicotine, the regression gave the result:

$$PM_{2.5} = 24.8 + 0.272T \text{ (N = 47; } R^2 = 0.40).$$

Thus each cigarette produces about a 0.3 (±0.03) $\mu g/m^3$ increase in the weekly mean $PM_{2.5}$ concentration, equivalent to a 2.1 (±0.2) $\mu g/m^3$ increase in the daily concentration.

Koutrakis et al. (1992) also analyzed the New York State data, using a mass-balance model to estimate $PM_{2.5}$ and elemental source strengths for cigarettes, wood burning stoves, and kerosene heaters. Homes with cigar or pipe smoking and fireplace use were eliminated, resulting in 178 indoor air samples. $PM_{2.5}$ source strength for smoking was estimated at 12.7 ± 0.8 (SE) mg/cigarette; but $PM_{2.5}$ source strengths could not be estimated for wood burning or kerosene heater usage (only seven homes in each category were available for analysis). For a residual category of all other indoor sources, a source strength of 1.16 mg/h was calculated. For nonsource homes (N = 49), the authors estimated that 60% (9 $\mu g/m^3$) of the total $PM_{2.5}$ mass was from outdoor sources and 40% (6 $\mu g/m^3$) from unidentified indoor sources. However, indoor concentrations were not significantly correlated with outdoor levels. For smoking homes, they estimated that 54% (26 $\mu g/m^3$) of the $PM_{2.5}$ mass was from smoking, 30% (15 $\mu g/m^3$) from outdoor sources, and 16% (8 $\mu g/m^3$) from unidentified sources. The elemental emissions profile for cigarettes included potassium (160 µg/cig), chlorine (69 µg/cig), and sulfur (65 µg/cig), as well as smaller amounts of bromine, cadmium, vanadium, and zinc. The woodburning profile included three elements: potassium (92 µg/h), silicon (44 µg/h) and calcium (38 µg/h). The kerosene heater profile included a major contribution from sulfur (1500 µg/h) and fairly large inputs of silicon (195 µg/h) and potassium (164 µg/h). A drawback of the mass-balance model was an inability to separately estimate the value of the penetration coefficient *P* and the decay rate *k* for particles and elements; Koutrakis et al. (1992) assumed a constant rate of 0.36 h^{-1} for *k*, and then solved for *P*.

7.2.2.1.3 The U.S. Environmental Protection Agency Particle Total Exposure Assessment Methodology Study

EPA designed a study of exposure to particles and associated elements in the late 1980s. Personal exposure and indoor and outdoor $PM_{2.5}$ and PM_{10} concentrations were measured. The personal exposure portion of the study is discussed in 7.4.1.1.1. The study was carried out under the Total Exposure Assessment Methodology (TEAM) program, and is known as the Particle TEAM, or PTEAM Study.

A pilot study was undertaken in nine homes in Azusa, CA in March of 1989 to test the sampling equipment. The first five households were monitored concurrently for seven days (March 6-13, 1989; Wiener, 1988, 1989; Wiener et al., 1990; Spengler et al., 1989); the last four households were then monitored for four consecutive days (March 16-20, 1989). Indoor and outdoor particle concentrations were monitored using impactors with a 10 Lpm pump (Marple et al., 1987). Indoor monitors, capable of sampling both fine and inhalable particles simultaneously, were placed in different rooms in each house to determine the magnitude of room-to-room variation.

Room-to-room variation of 12-h integrated particle levels was generally less than 10%. Therefore the several indoor values in a particular house were averaged to provide a single mean indoor value to compare to the corresponding outdoor value. The mean (SE) 24-h indoor PM_{10} concentration was 58.7 (3.4) $\mu g/m^3$ compared to the outdoor mean of 62.6 (3.5) $\mu g/m^3$. Corresponding $PM_{2.5}$ concentrations were 36.3 (2.6) $\mu g/m^3$ indoors and 42.6 (3.0) $\mu g/m^3$ outdoors.

Regressions of indoor on outdoor concentrations (N = 26 for each size fraction and time period) resulted in the following equations for PM_{10}:

$$C_{in}\ (\text{day}) = 36\ (11) + 0.44\ (0.14)\ C_{out} \quad (R^2 = 0.17)$$
$$C_{in}\ (\text{night}) = 44\ (11) + 0.14\ (0.19)\ C_{out} \quad (R^2 = 0.01)$$

and for $PM_{2.5}$:

$$C_{in}\ (\text{day}) = 18\ (5) + 0.47\ (0.10)\ C_{out} \quad (R^2 = 0.30)$$
$$C_{in}\ (\text{night}) = 24\ (6) + 0.23\ (0.15)\ C_{out} \quad (R^2 = 0.05)$$

where the values in parentheses are the standard errors of the parameter estimates. (In most epidemiology studies, PM exposures are related to PM concentrations at a community ambient monitoring station, rather than to these PM concentrations measured outside indivdual homes).

The R^2 values improved considerably when the regressions for individual homes were calculated (Wallace, 1996; see also Table 7-6). For the five homes with seven days of monitoring (14 12-h periods) all slopes were significant, and R^2 values ranged from 0.34 to 0.79 for PM_{10} and from 0.49 to 0.85 for $PM_{2.5}$. For the four homes having only four days of monitoring, only home 8 had significant slopes and R^2 values above 0.5.

TABLE 7-6. REGRESSIONS OF INDOOR ON OUTDOOR PM_{10} AND $PM_{2.5}$ CONCENTRATIONS ($\mu g/m^3$): PARTICLE TOTAL EXPOSURE ASSESSMENT METHODOLOGY PREPILOT STUDY

PM_{10} ($\mu g/m^3$)								
House	N	Intercept	SE	p	Slope	SE	p	R^2
1	13	23	9	0.026	0.27	0.12	0.038	0.34
2	13	−25	17	NS	1.14	0.23	0.0003	0.7
3	14	13	7	NS	0.64	0.1	0.00002	0.79
4	13	16	9	NS	0.52	0.14	0.004	0.54
5	14	14	13	NS	0.67	0.16	0.001	0.59
6	8	175	38	0.004	−1.52	0.78	NS	0.39
7	8	30	34	NS	0.34	0.62	NS	0.05
8	8	−2.7	23	NS	1.38	0.5	0.03	0.56
9	7	48	42	NS	0.94	0.87	NS	0.19
$PM_{2.5}$ ($\mu g/m^3$)								
House	N	Intercept	SE	p	Slope	SE	p	R^2
1	14	14	3.4	0.001	0.19	0.06	0.005	0.49
2	14	−12	9	NS	0.96	0.16	0.00007	0.74
3	14	7.3	4.5	NS	0.72	0.09	0.00001	0.85
4	13	6	5	NS	0.52	0.13	0.002	0.6
5	14	11	6	NS	0.58	0.1	0.0001	0.72
6	8	65	26	0.046	−0.32	1.01	NS	0.02
7	8	10	8	NS	0.35	0.22	NS	0.3
8	8	−0.34	13	NS	0.99	0.39	0.045	0.51
9	8	37	47	NS	0.78	1.3	NS	0.05

Source: Data from PTEAM Prepilot Study upon which R^2 values were generated as reported by Wallace (1996).

After the pilot study in Azusa, CA, the EPA sponsored a study of personal, indoor, and outdoor concentrations of PM_{10}, and indoor and outdoor concentrations of $PM_{2.5}$ in Riverside, CA (Pellizzari et al., 1992, 1993; Perritt et al., 1991; Sheldon et al., 1992; Clayton et al., 1993; Thomas et al., 1993; Özkaynak et al., 1993a,b, 1996). Personal exposure results of this study are discussed in Section 7.4.1.1.2. The main goal was to estimate the frequency distribution of exposures to PM_{10} for all nonsmoking Riverside residents aged 10 and above; and 178 households were selected, using probability sampling to represent about 61,000 households throughout most of the city of Riverside. Homes were sampled between September 22 and November 9, 1990, and each home had two 12-h samples for both size fractions. A central site operated throughout the 48 days of the study, producing 96 12-h samples collected by side-by-side reference samplers (dichotomous samplers and modified hi-volume samplers) along with the low-flow (4 Lpm) impactors with nominal cutpoints at 2.5 and 10 μm designed for this study. (Laboratory tests [Thomas et al., 1993] revealed that the actual cutpoints were 2.5 μm and 11.0 μm, but this section shall refer to PM_{10} in keeping with the investigators [Clayton et al., 1993] who reported their data as PM_{10}). A subset of the homes was monitored for PAHs (Sheldon et al., 1992); 125 were monitored indoors and 65 of those were monitored outdoors for two consecutive 12-h periods.

The precision of the three types of particle samplers at the central site was excellent, with median RSDs of about 4 to 5% (Wallace, et al., 1991a). The low-flow sampler produced estimates about 12% greater than the dichotomous sampler, which was about 7% greater than the modified hi-vol sampler (Wallace, et al., 1991b). Part of the difference may be due to the different cutpoints (estimated to be 11 μm for the new sampler, 9.5 for the dichot, and 9.0 for the modified hi-vol), and part due to particle bounce (large particles bouncing off the impactor and being re-entrained in the flow to the filter), such that the $PM_{2.5}$ and PM_{10} fractions in the low-flow sampler may be contaminated with a small number of larger-size particles. However, particle bounce was found in laboratory tests to account for less than 7% of the total mass.

The population-weighted distributions of personal (PEM), indoor (SIM), and outdoor (SAM) particle concentrations are provided in Table 7-7. PM_{10} mean concentrations (150 $\mu g/m^3$) were more than 50% higher than either indoor or outdoor levels (95 $\mu g/m^3$).

TABLE 7-7. WEIGHTED DISTRIBUTIONS OF PERSONAL, INDOOR, AND OUTDOOR[a] PARTICLE CONCENTRATIONS ($\mu g/m^3$)

	DAYTIME					NIGHTTIME				
	$PM_{2.5}$		PM_{10}			$PM_{2.5}$		PM_{10}		
	SAM	SIM	SAM	SIM	PEM	SAM	SIM	SAM	SIM	PEM
Sample size	167	173	165	169	171	161	166	162	163	168
Minimum	7.4	2.8	16.2	16.6	35.1	3.4	2.9	13.6	14.1	19.1
Maximum	187.8	238.3	506.6	512.8	454.8	164.2	133.3	222.9	180.3	278.3
Mean	48.9	48.2	94.9	94.7	149.8	50.5	36.2	86.3	62.7	76.8
(Std. error)	(3.5)	(4.1)	(5.5)	(5.7)	(9.2)	(3.7)	(2.2)	(4.4)	(3.2)	(3.5)
Geometric Mean	37.7	35.0	82.7	78.2	128.7	37.2	26.7	74.5	53.1	67.9
(Std. error)	(2.5)	(3.3)	(4.1)	(5.0)	(8.5)	(3.1)	(1.9)	(4.0)	(3.1)	(3.1)
Std. deviation	37.6	41.2	57.2	61.4	84.3	40.3	29.5	47.7	37.4	39.7
Geometric std. deviation[b]	2.07	2.25	1.68	1.88	1.75	2.23	2.21	1.74	1.78	1.64
Percentiles										
10th	14.9	11.5	42.8	30.9	59.9	14.5	10.0	39.3	25.2	36.6
25th	23.4	19.3	56.9	49.5	86.1	23.0	14.8	53.6	33.5	48.1
50th (median)	35.5	33.5	84.1	81.7	129.7	35.0	25.9	74.1	51.6	66.2
75th	60.1	61.5	110.8	127.2	189.1	64.9	48.9	103.7	84.8	98.8
90th	102.2	101.0	157.2	180.7	263.1	120.7	82.7	167.8	116.9	135.0
Std. errors of percentiles										
10th	1.6	3.4	2.3	3.4	4.0	2.1	0.9	7.4	1.5	1.5
25th	2.1	1.4	4.5	4.3	9.4	2.7	1.3	3.4	2.4	3.1
50th	4.0	4.5	4.7	8.3	7.5	2.4	2.4	4.8	3.5	4.3
75th	3.9	3.3	4.0	9.4	10.8	4.6	5.3	5.1	4.7	8.2
90th	4.6	6.7	7.2	11.0	12.0	5.8	5.8	4.3	5.3	10.1

[a]Statistics other than the sample size, minimum, and maximum are calculated using weighted data; they provide estimates for the target population of person-days (PEM) or of household-days (SIM, SAM).
[b]In contrast to the other statistics, the gsd is a unitless quantity.

Source: Pellizzari et al. (1992).

Overnight mean personal PM_{10} concentrations (77 $\mu g/m^3$) were similar to the indoor (63 $\mu g/m^3$) and outdoor (86 $\mu g/m^3$) levels. The reason for the higher daytime personal exposures (PEM) than daytime SIM or SAM is not completely understood: it may be due to persons often being close to sources of particles (e.g., cooking, dusting, or vacuuming) or to re-entrainment of household dust (Thatcher and Layton, 1995). It appears not to be due to skin flakes or clothing fibers; many skin flakes were found on filters but their mass does not account for more than 10% of the excess personal exposure (Mamane, 1992).

Mean $PM_{2.5}$ daytime concentrations were similar indoors (48 $\mu g/m^3$) and outdoors (49 $\mu g/m^3$), but indoor concentrations fell off during the sleeping period (36 $\mu g/m^3$) compared to 50 $\mu g/m^3$ outdoors. Thus the fine particle contribution to PM_{10} concentrations averaged about 51% during the day and 58% at night, both indoors and outdoors. The distributions of these ratios are provided in Table 7-8.

TABLE 7-8. WEIGHTED DISTRIBUTIONS[a] OF $PM_{2.5}/PM_{10}$ CONCENTRATION RATIO

	Daytime		Nighttime	
	Outdoor	Indoor	Outdoor	Indoor
Sample Size	160	167	154	160
Mean	0.470	0.492	0.522	0.550
(Std. error)	(0.016)	(0.021)	(0.017)	(0.014)
Geometric Mean	0.444	0.455	0.497	0.517
(Std. error)	(0.017)	(0.022)	(0.019)	(0.016)
Percentiles				
10th	0.274	0.250	0.308	0.301
25th	0.371	0.347	0.406	0.440
50th (median)	0.469	0.498	0.515	0.556
75th	0.571	0.607	0.646	0.694
90th	0.671	0.735	0.731	0.771
Std. errors of percentiles				
10th	0.018	0.030	0.023	0.023
25th	0.018	0.046	0.028	0.017
50th	0.015	0.020	0.022	0.015
75th	0.019	0.024	0.027	0.023
90th	0.012	0.028	0.016	0.012

[a]Statistics other than sample size are calculated using weighted data; they provide estimates for the target population of household-days.

Source: Pellizzari et al. (1992).

Unweighted distributions are displayed in Figures 7-6 and 7-7 for 24-h average PM_{10} and $PM_{2.5}$ personal, indoor, and outdoor concentrations. For 24-h data, the indoor PM is less than the outdoor PM at all percentiles. Most of the distributions were not significantly different from log-normal distributions, as determined by a chi-square test. About 25% of the nonsmoking population of Riverside was estimated to have 24-h personal PM_{10} exposures exceeding the 150 $\mu g/m^3$ 24-h NAAQS for ambient air. Since participants were monitored for only one day, the percentage of persons with exposures exceeding the outdoor 24-h standard more than once per year would be greater than 25%.

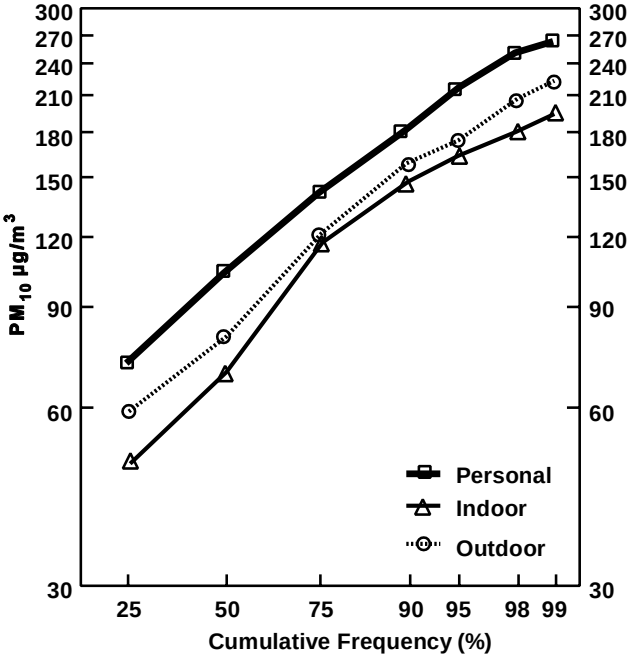


Figure 7-6. Cumulative frequency distribution of 24-h personal, indoor, and outdoor PM_{10} concentrations in Riverside, CA.

Source: Adapted from PTEAM study data (Pellizzari et al., 1992).

The 48-day sequence of outdoor PM_{10} and $PM_{2.5}$ concentrations is shown in Figure 7-8 (Wallace et al., 1991a). At least two extended episodes of high fine-particle concentrations occurred, and four days of high Santa Ana winds, with correspondingly high coarse-particle concentrations from desert sand, were observed.

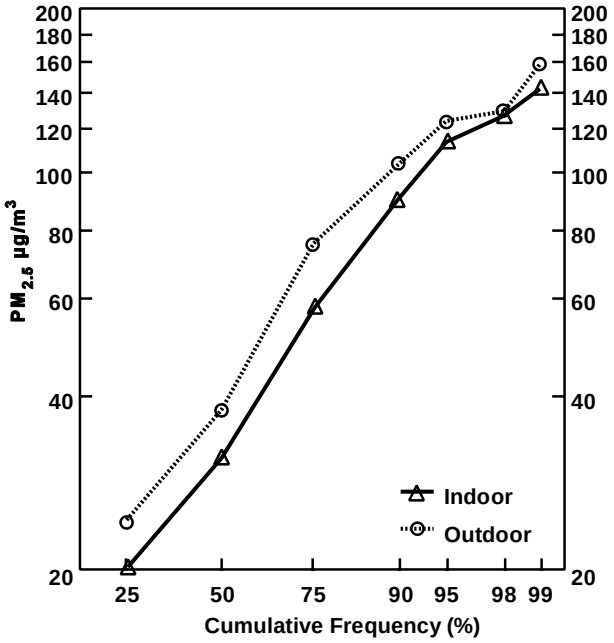


Figure 7-7. Cumulative frequency distribution of 24-h indoor and outdoor $PM_{2.5}$ concentrations in Riverside, CA.

Source: Adapted from PTEAM study data (Pellizzari et al., 1992).

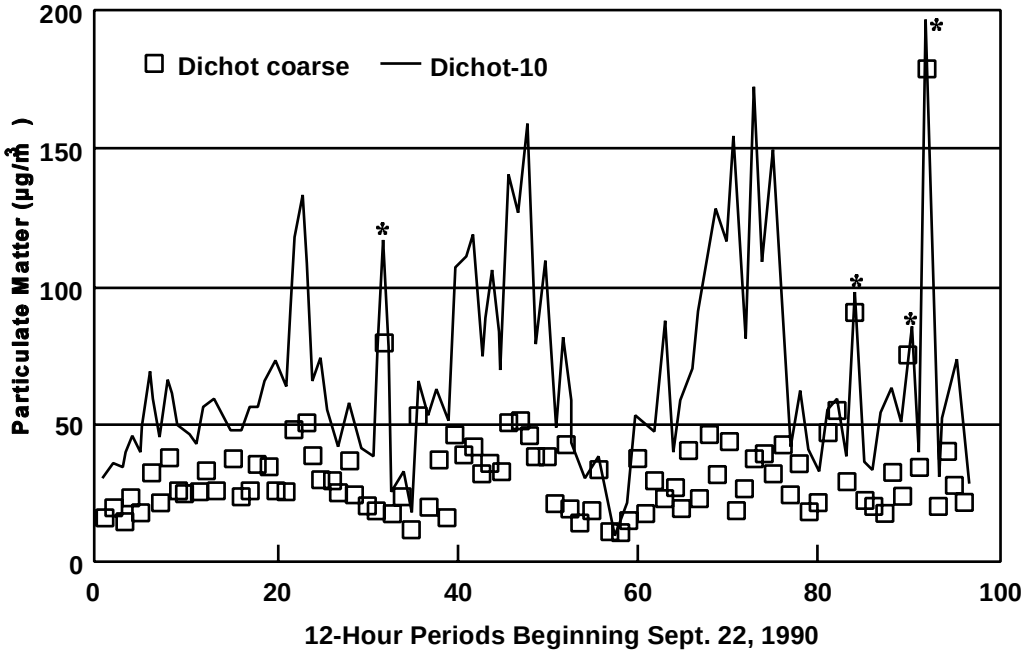


Figure 7-8. Forty-eight day sequence of PM_{10} and coarse PM (PM_{10} – $PM_{2.5}$) in Riverside, CA, PTEAM study. Santa Ana wind conditions are noted by an asterisk.

Source: Wallace et al. (1991a).

Central-site $PM_{2.5}$ and PM_{10} concentrations agreed well with back yard concentrations. Pearson correlations of the log-transformed data were 0.96 and 0.92 for overnight and daytime $PM_{2.5}$ and 0.93 for overnight PM_{10} values (Özkaynak et al., 1993a), but dropped to 0.64 for daytime PM_{10} values. However, two homes in one Riverside area showed very high outdoor concentrations of 380 and 500 $\mu g/m^3$ on one day, while two homes in another Riverside area and the central-site monitor showed more typical concentrations. A local event likely produced the higher concentrations at the former two homes. If they are removed from the data set, the correlation improves from 0.64 to 0.90, suggesting that a single central-site monitor can represent well $PM_{2.5}$ and PM_{10} concentrations throughout a wider area such as a town or small city (at least in the Riverside area) except for unusual local conditions.

Daytime indoor PM_{10} and $PM_{2.5}$ concentrations showed low-to-moderate Pearson correlations of 0.46 and 0.55, respectively, with outdoor concentrations (N = 158 to 173). At night, the correlations improved somewhat to 0.65 and 0.61, respectively (N = 50 to 168). Outdoor PM_{10} concentrations explained about 27% of the variance of indoor levels (Figure 7-9) with the two outliers included.

Simple regressions of indoor on outdoor PM_{10} and $PM_{2.5}$ resulted in the following equations (standard errors in parentheses):

Indoor PM_{10} = 48 (9) + 0.51 (0.08) × Outdoor PM_{10} (day)	N=159	R^2 = 0.22
Indoor PM_{10} = 20 (5) + 0.52 (0.05) × Outdoor PM_{10} (night)	N=151	R^2 = 0.42
Indoor $PM_{2.5}$ = 14 (4) + 0.70 (0.07) × Outdoor $PM_{2.5}$ (day)	N=162	R^2 = 0.42
Indoor $PM_{2.5}$ = 9 (3) + 0.56 (0.04) × Outdoor $PM_{2.5}$ (night)	N=153	R^2 = 0.54

Simple regressions of personal PM_{10} on outdoor and indoor PM_{10} resulted in the following equations:

Personal PM_{10} = 71 (9) + 0.78 (0.08) × Indoor PM_{10} (day)	N=163	R^2 = 0.40
Personal PM_{10} = 21 (4) + 0.90 (0.05) × Indoor PM_{10} (night)	N=158	R^2 = 0.65
Personal PM_{10} = 100 (12) + 0.48 (0.10) × Outdoor PM_{10} (day)	N=158	R^2 = 0.12
Personal PM_{10} = 31 (6) + 0.53 (0.06) × Outdoor PM_{10} (night)	N=155	R^2 = 0.38

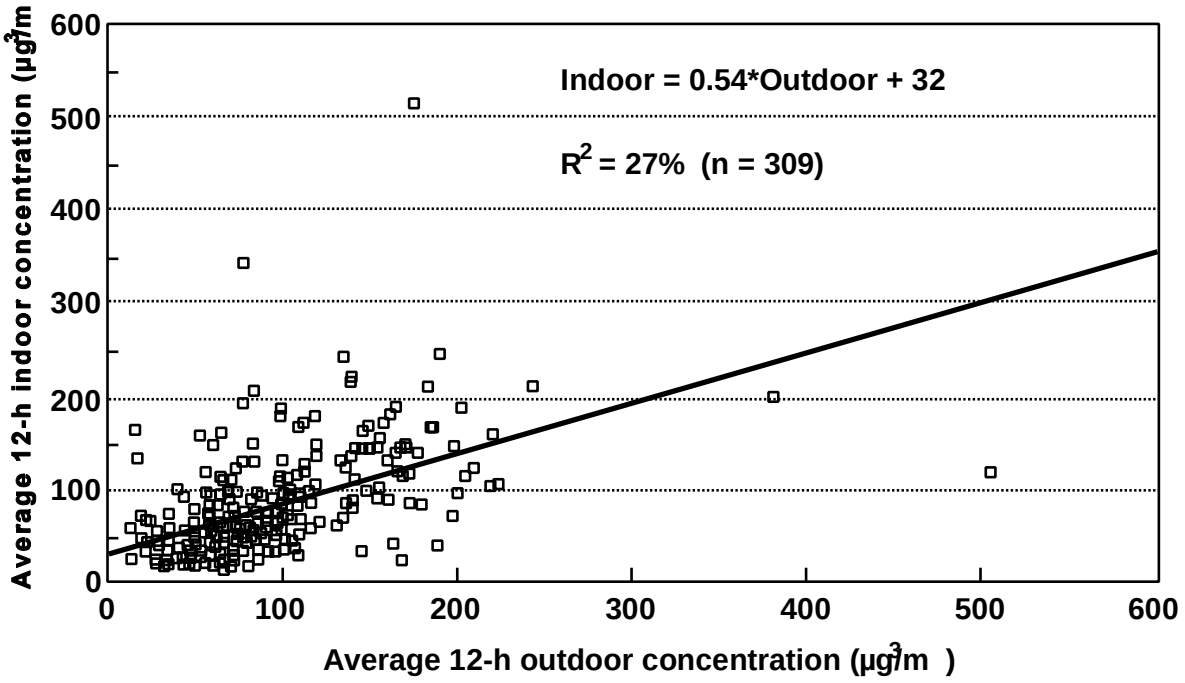


Figure 7-9. Average indoor and outdoor 12-h concentrations of PM_{10} during the PTEAM study in Riverside, CA.

Source: Özkaynak et al. (1993b).

Correlation analyses and regressions relating personal to indoor, indoor to outdoor, and personal to outdoor concentrations of the 14 prevalent elements were carried out for the appropriate size fractions and both 12-h monitoring periods. For most of the elements, as with particle mass, moderate correlations were noted for personal-indoor and indoor-outdoor concentrations but low correlations for personal-outdoor concentrations. One element was a strong exception to this rule: sulfur. Unlike any of the other elements, sulfur was not elevated in the PEM relative to the SIM, and, thus, personal concentrations were much more closely related to indoor concentrations (r_s = 0.91 during the day and 0.95 at night). Moreover, because few sources of sulfur are found indoors, the indoor-outdoor correlations were high (r_s varied between 0.90 and 0.95 for both size fractions), and even the personal-outdoor correlations showed little degradation (the Spearman correlation r_s = 0.85 during the day and 0.92 at night).

Regressions of outdoor sulfur on indoor levels gave the following results for PM_{10} sulfur ($\mu g/m^3$):

S_{in} (day) = 0.26 (0.06 SE) + 0.80 (0.02) S_{out} N = 164 R^2 = 0.88

S_{in} (night) = 0.20 (0.06) + 0.71 (0.03) S_{out} N = 155 R^2 = 0.84

and for fine ($PM_{2.5}$) sulfur:

S_{in} (day) = 0.046 (0.04 SE) + 0.85 (0.02) S_{out} N = 164 R^2 = 0.92

S_{in} (night) = 0.061 (0.04) + 0.80 (0.02) S_{out} N = 154 R^2 = 0.89

Stepwise regressions resulted in smoking, cooking, and either air exchange rates or house volumes being added to outdoor concentrations as significant variables (Table 7-9). Homes with smoking added about 27 to 32 µg/m^3 to the total $PM_{2.5}$ concentrations and about 29 to 37 μg/m^3 to the PM_{10} values. Cooking added 12 to 26 µg/m^3 to the daytime PM_{10} concentration and about 13 μg/m^3 to the daytime $PM_{2.5}$ concentration, but was not significant during the overnight period.

A model developed by Koutrakis et al. (1992) was solved using nonlinear least squares to estimate penetration factors, decay rates, and source strengths for particles and elements from both size fractions in the PTEAM study. In this model, which assumes perfect instantaneous mixing and steady-state conditions throughout each 12-h monitoring period, the indoor concentration of particles or elements is given by

$$C_{in} = \frac{PaC_{out} + Q_{is}/V}{a + k} \qquad (7\text{-}3)$$

where

C_{in} = indoor concentration (ng/m^3 for elements, µg/m^3 for particles)
P = penetration coefficient
a = air exchange rate (h^{-1})
C_{out} = outdoor concentration (ng/m^3 or µg/m^3)
Q_{is} = mass flux generated by indoor sources (ng/h or µg/h)
V = volume of room or house (m^3)
k = decay rate due to diffusion or sedimentation (h^{-1})

From initial multivariate analyses, the most important indoor sources appeared to be smoking and cooking. Therefore the indoor source term Q_{is} was replaced by the following expression:

TABLE 7-9. STEPWISE REGRESSION RESULTS FOR INDOOR AIR CONCENTRATIONS OF PM_{10} AND $PM_{2.5}$ ($\mu g/m^3$) COEFFICIENTS (STANDARD ERRORS OF ESTIMATES)

	PM_{10}			$PM_{2.5}$		
Variable	All	Day	Night	All	Day	Night
N	310	158	147	324	156	149
R^2	41%	39%	58%	55%	53%	71%
Intercept		57			21	
		(21)			(7.8)	
Outdoor air	0.52	0.66	0.45	0.64	0.71	0.53
	(0.05)	(0.09)	(0.05)	(0.04)	(0.07)	(0.04)
Smoking[a]	37	29	38	28	27	32
	(6)	(8)	(11)	(3.5)	(7)	(10)
No. cigarettes[b]	3.2	3.0	3.9	2.5	2.4	4.0
	(0.7)	(1.0)	(0.9)	(0.4)	(0.6)	(0.6)
Cooking[c]	20	26	12	9.4	13	
	(5)	(9)	(5)	(2.9)	(5)	
Air exchange	5.2		12			4.5
	(2.0)		(5)			(2)
House volume[d]	−0.08	−2.7			−2.0	
	(0.02)	(1)			(0.6)	

All listed coefficients significantly different from zero at $p < 0.05$.
[a]Binary variable: 1 = at least one cigarette smoked in home during monitoring period.
[b]This variable was interchanged with the smoking variable in alternate regressions to avoid colinearity problems.
[c]Binary variable: 1 = cooking reported for at least one min in home during monitoring period.
[d]Volume in thousands of cubic feet.

Source: Özkaynak et al. (1996).

$$Q_{is} = (N_{cig}S_{cig} + T_{cook}S_{cook})/T + Q_{other} \quad (7\text{-}4)$$

where

T = duration of the monitoring period (h)
N_{cig} = number of cigarettes smoked during monitoring period
S_{cig} = mass of elements or particles generated per cigarette smoked (ng/cig or µg/cig)
T_{cook} = time spent cooking (min) during monitoring period
S_{cook} = mass of elements or particles generated per min of cooking (ng/min or µg/min)
Q_{other} = mass flux of elements or particles from all other indoor sources (ng/h or µg/h)

With these changes, the equation for the indoor concentration due to these indoor sources becomes

$$C_{in} = \frac{PaC_{out}}{a+k} + \frac{N_{cig}S_{cig} + T_{cook}S_{cook}}{(a+k)V\,T} + \frac{Q_{other}}{(a+k)V} \qquad (7\text{-}5)$$

The indoor and outdoor concentrations, number of cigarettes smoked, monitoring duration, time spent cooking, house volumes, and air exchange rates were all measured or recorded. The penetration factor, decay rates, and source strengths for smoking, cooking, and all other indoor sources (Q_{other}) were estimated using a nonlinear model (NLIN in SAS software). The Gauss-Newton approximation technique was used to regress the residuals onto the partial derivatives of the model with respect to the unknown parameters until the estimates converge. On the first run, the penetration coefficients were allowed to "float" (no requirement was made that they be ≤ 1). Since nearly all coefficients came out close to 1, a second run was made bounding them from above by 1. The NLIN program provides statistical uncertainties (upper and lower 95% confidence intervals) for all parameter estimates. However, it should be noted that these uncertainties assume perfect measurements and are therefore underestimates of the true uncertainties.

Results are presented in Table 7-10 for the combined day and night samples. The penetration factors were very close to unity for nearly all particles and elements. The calculated average decay rate (lower and upper 95% confidence levels) for $PM_{2.5}$ was 0.39 (0.22; 0.55) h^{-1}, and for PM_{10} was 0.65 (0.36; 0.93) h^{-1}. Since PM_{10} contains the $PM_{2.5}$ fraction, a separate calculation was made for the coarse particles ($PM_{10} - PM_{2.5}$) with a resulting decay rate of 1.01 (0.6; 1.4) h^{-1}. Each cigarette emitted 22 (14; 30) mg of PM_{10} on average, about two-thirds of which 14 (10; 17) mg is in the fine fraction. Cooking emitted 4.1 (2.6; 5.7) mg/min of inhalable particles, of which about 40% or 1.7 (1.0; 2.3) mg/min, was in the fine fraction. All target elements emitted by cooking were limited almost completely to the coarse fraction. Sources other than cooking and smoking emitted about 5.6 (2.6; 8.7) mg/h of PM_{10}, of which only about 1.1 mg/h (0.0; 2.1) (20%) was in the fine fraction.

Decay rates for elements associated with the fine fraction were generally lower than for elements associated with the coarse fraction, as would be expected. For example, sulfur,

TABLE 7-10. PENETRATION FACTORS, DECAY RATES, AND SOURCE STRENGTHS: NONLINEAR ESTIMATES

	Penetration			Decay Rate (1/h)			S_cook (μg/min)			S_smoke (μg/cig)			Other Sources (μg/h)		
VAR	Mean	l95	u95	Mean	l95	u95	Mean	l95	u95	Mean [b]	l95	u95	Mean [b]	l95	u95
$PM_{2.5}$ [a]	1.00	0.89	1.11	0.39	0.22	0.55	1.7	1.0	2.3	13.8	10.2	17.3	1.1	0.0	2.1
Al	1.00	0.95	1.05	0.03	−0.03	0.09	0.9	−1.4	3.1	9.0	−2.5	20.5	3.0	−3.7	9.8
Mn	0.87	0.78	0.95	0.23	0.07	0.38	0.1	−0.1	0.2	0.2	−0.4	0.8	0.5	0.2	0.9
Br	0.90	0.81	0.99	0.28	0.15	0.41	0.1	0.0	0.2	1.9	1.3	2.5	0.6	0.3	0.9
Pb	Fail to converge														
Ti	Fail to converge														
Cu	1.00	0.56	1.44	1.63	0.38	2.88	0.6	0.0	1.2	3.7	0.2	7.2	3.8	1.4	6.3
Sr	0.97	0.93	1.01	0.07	0.01	0.12	0.0	0.0	0.0	0.1	−0.1	0.2	0.1	0.0	0.2
Si	0.98	0.75	1.20	0.54	0.04	1.05	6.1	−8.6	20.9	14.4	−58.3	87.2	57.3	12.5	102.0
Ca	1.00	0.65	1.35	0.61	−0.02	1.25	11.9	−0.6	24.4	165.6	72.0	259.1	34.1	3.4	64.8
Fe	1.00	0.76	1.24	0.70	0.11	1.29	4.5	−3.3	12.3	23.8	−16.3	63.9	23.8	1.8	45.7
K	1.00	0.81	1.19	0.16	−0.04	0.37	0.0	−4.4	4.4	121.3	85.7	156.9	8.9	−0.5	18.3
S	1.00	0.97	1.03	0.16	0.12	0.19	1.0	−3.9	5.9	27.1	2.4	51.7	4.0	−3.7	11.7
Zn	0.71	0.57	0.86	0.78	0.31	1.25	0.4	−0.5	1.2	2.9	−1.5	7.4	7.5	4.2	10.9
Cl	0.50	0.28	0.72	0.64	0.05	1.24	5.9	0.1	11.6	102.6	54.0	151.2	20.6	7.2	34.0
PM_{10} [a]	1.00	0.85	1.15	0.65	0.36	0.93	4.1	2.6	5.7	21.9	13.6	30.2	5.6	2.6	8.7
Al	1.00	0.80	1.20	0.80	0.38	1.21	69.5	16.6	122.4	97.6	−159.0	354.2	154.5	52.0	257.0
Mn	1.00	0.80	1.20	0.69	0.30	1.07	0.9	0.1	1.7	1.1	−2.7	4.9	1.2	−0.2	2.6
Br	1.00	0.90	1.10	0.21	0.11	0.32	0.1	0.0	0.3	1.8	1.2	2.5	0.4	0.1	0.6
Pb	1.00	0.89	1.11	0.14	0.01	0.26	0.0	−0.3	0.3	2.1	0.4	3.9	0.0	−0.6	0.6
Ti	1.00	0.80	1.20	0.60	0.22	0.98	4.0	0.3	7.8	10.0	−8.4	28.4	10.3	2.6	18.1
Cu	0.83	0.62	1.05	0.77	0.18	1.36	0.5	0.0	1.1	3.5	0.4	6.5	3.2	1.3	5.1
Sr	1.00	0.83	1.16	0.62	0.28	0.97	0.3	0.0	0.5	2.6	1.2	3.9	0.9	0.3	1.5
Si	1.00	0.81	1.19	0.62	0.26	0.97	149.3	26.9	271.8	296.4	−293.9	886.6	237.8	16.1	459.6
Ca	1.00	0.68	1.32	0.63	0.06	1.20	118.7	37.3	200.1	800.0	329.0	1271.0	107.6	−27.0	242.3
Fe	1.00	0.80	1.20	0.66	0.26	1.06	46.7	8.5	84.8	73.0	−109.8	255.9	51.5	−15.5	118.5
K	1.00	0.83	1.17	0.46	0.17	0.75	17.6	0.1	35.2	215.7	116.9	314.5	43.6	8.6	78.5
S	1.00	0.96	1.04	0.21	0.17	0.26	6.8	−0.7	14.3	68.0	29.3	106.7	22.7	10.4	34.9
Zn	1.00	0.81	1.19	0.37	0.10	0.64	1.2	−0.2	2.5	4.0	−3.0	11.0	7.4	3.4	11.4
Cl	0.94	0.44	1.43	2.36	0.48	4.24	45.7	17.6	73.9	320.2	107.0	533.4	148.4	49.4	247.4

[a]Mass units in mg for $PM_{2.5}$ and PM_{10} only.
[b]A negative lower confidence interval implies a nonzero mean is not statistically significant.

Source: Özkaynak et al. (1993a).

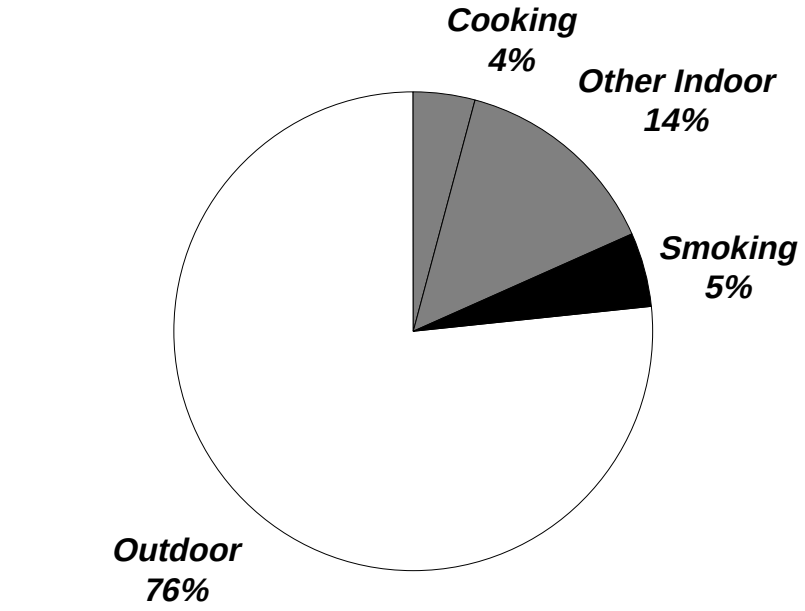
which has the lowest mass median diameter of all the elements, had calculated decay rates of 0.16 (0.12; 0.19) h^{-1} and 0.21 (0.17; 0.26) h^{-1} for $PM_{2.5}$ and PM_{10} fractions, respectively. The crustal elements (Ca, Al, Mn, Fe) had decay rates ranging from 0.6 to 0.8 h^{-1}.

Based on the mass-balance model, outdoor air was the major source of indoor particles, providing about 3/4 of fine particles and 2/3 of thoracic particles in the average home. It was also the major source for most of the target elements, providing 70 to 100% of the observed indoor concentrations for 12 of the 15 elements. It should be noted that these conclusions are applicable only to Riverside, CA. In five of the six cities studied by Harvard and in both New York counties, outdoor air could not have provided as much as half of the indoor air particle mass for the average home, because the observed indoor-outdoor ratios of the mean concentrations were ≥ 2. However, for homes without smoking or combustion sources (Santanam et al., 1990; Leaderer et al., 1990; Table 7-5), indoor-outdoor ratios were ≈ 1. In general, homes in areas with colder winters (such as New York) would be expected to have tighter construction than homes in warmer areas (such as Riverside) and, therefore, more protection against outdoor air particles.

Unidentified indoor sources accounted for most of the remaining particle and elemental mass collected on the indoor monitors. The nature of these sources is not yet completely understood (Thatcher and Layton, 1995). They apparently do not include smoking, other combustion sources, cooking, dusting, vacuuming, spraying, or cleaning, since all these sources together account for less than the unidentified sources. For example, the unidentified sources accounted for 26% of the average indoor PM_{10} particles, whereas smoking accounted for 4% and cooking for 5% (Figure 7-10).

Of the identified indoor sources, the two most important were smoking and cooking (Figures 7-11 and 7-12). Smoking was estimated to increase 12-h average indoor concentrations of PM_{10} and $PM_{2.5}$ by 3.2 and 2.5 $\mu g/m^3$ per cigarette, respectively. Homes with smokers averaged about 30 $\mu g/m^3$ higher levels of PM_{10} than homes without smokers, most of this increase being in the fine fraction. Cooking increased indoor concentrations of PM_{10} by about 0.6 $\mu g/m^3$ per minute of cooking, most of the increase being in coarse particles.

Emission profiles for target elements were obtained for smoking and for cooking. Major elements emitted by cigarettes were K, Cl, and Ca; those from cooking included Al,



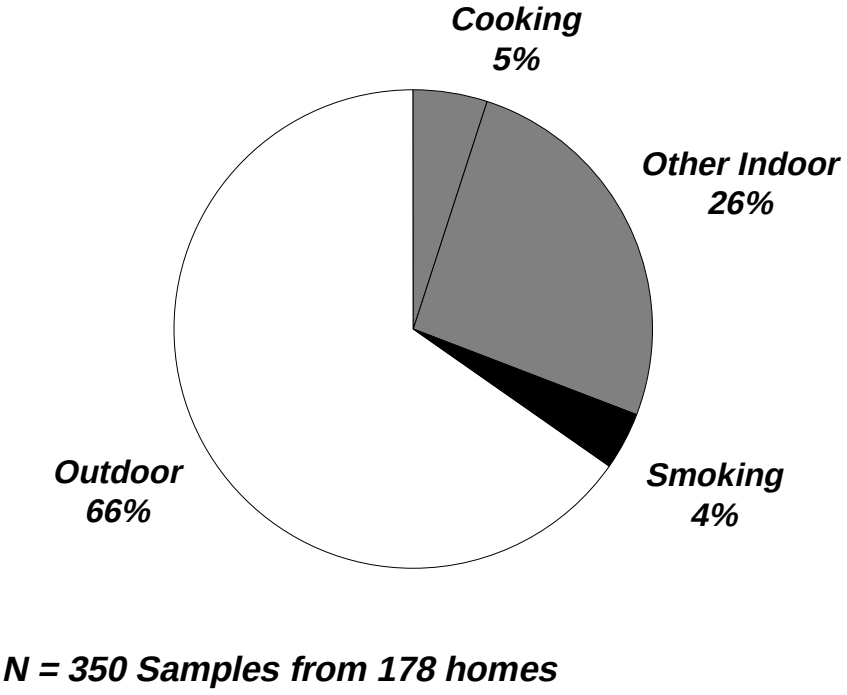
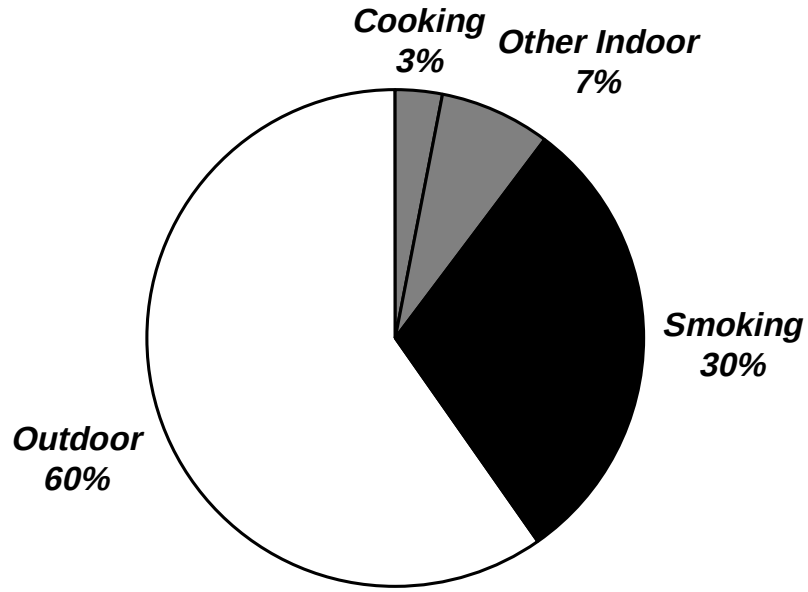


Figure 7-10. Sources of fine particles ($PM_{2.5}$) (top) and thoracic particles (PM_{10}) (bottom) in all homes (Riverside, CA).

Source: Özkaynak et al. (1993a).



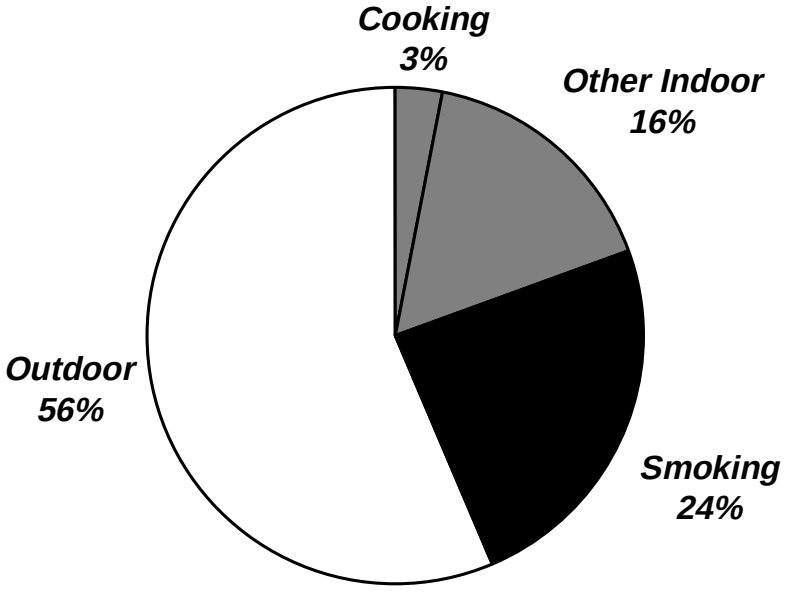
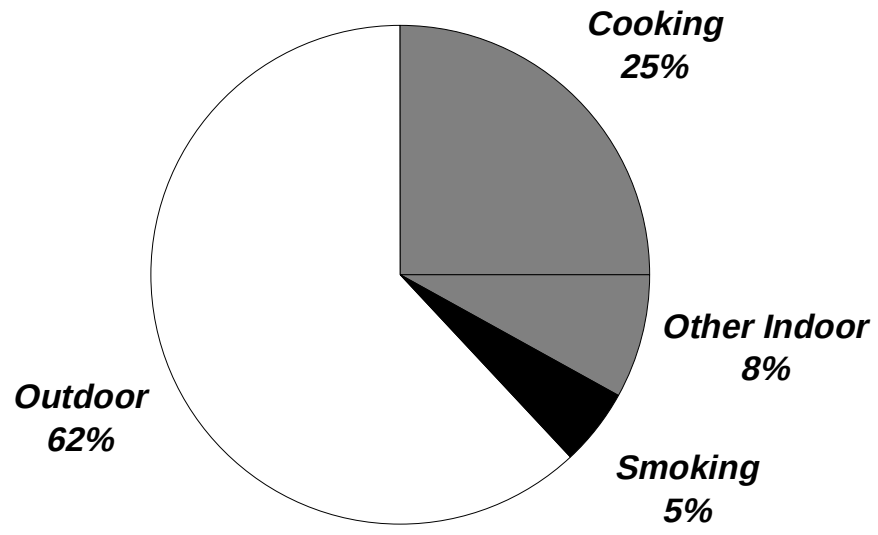


Figure 7-11. Sources of fine particles ($PM_{2.5}$) (top) and thoracic particles (PM_{10}) (bottom) in homes with smokers (Riverside, CA).

Source: Özkaynak et al. (1993a).



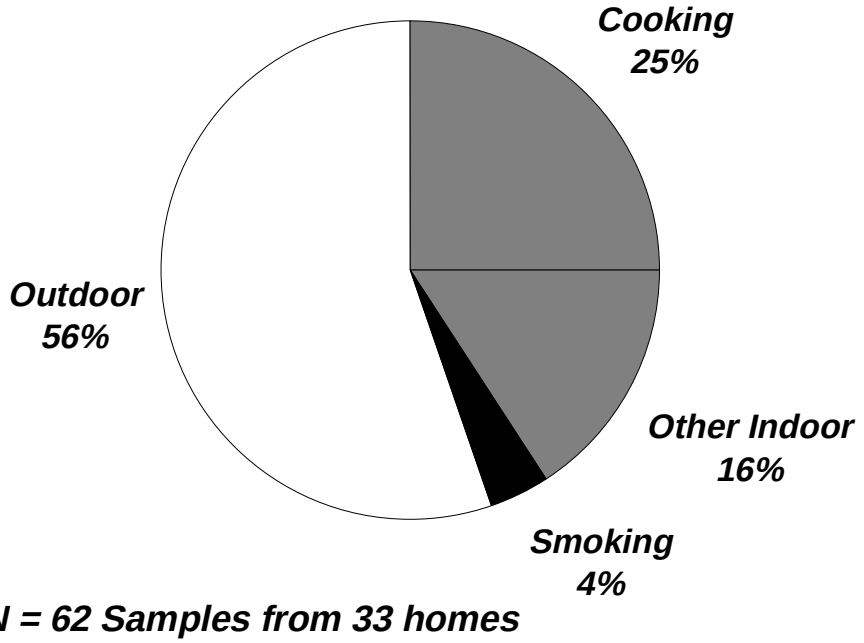


Figure 7-12. Sources of fine particles ($PM_{2.5}$) and thoracic particles (PM_{10}), top and bottom panels, respectively, for homes with cooking during data collection (Riverside, CA).

Source: Özykaynak et al. (1993a).

Fe, Ca, and Cl. Other household activities such as vacuuming and dusting appeared to make smaller contributions to indoor particle levels. Commuting and working outside the home resulted in lower particle exposures than for persons staying at home. As with the particle mass, daytime personal exposures to 14 of 15 elements were consistently higher than either indoor or outdoor concentrations. At night, levels of the elements were similar in all three types of samples.

7.2.2.1.4 Comparison of the Three Large-Scale Studies

The three studies had somewhat different aims and therefore different study designs. The Harvard Six-City study selected homes based on various criteria, especially a requirement that a school-age child be in the home, but did not employ a probability-based sample. Therefore the results strictly apply only to the homes in the sample and not to a wider population; however, the very large number of homes suggests that the results should be broadly applicable to homes with school-age children in the six cities. The New York State study used a probability-based sample, but stratified on the basis of combustion sources. Hence, there are likely to be a higher fraction of homes with kerosene heaters, wood stoves, and fireplaces in the sample than in the general population. The PTEAM study used a fully probability-based procedure, and its results are likely the most broadly applicable to the entire population of Riverside households. However, the participants were limited to nonsmokers, so homes with only smokers were excluded; as a consequence, maximum indoor concentrations were likely underestimated. Also, the three studies used different monitors, with different cutpoints precluding exact comparisons. However, large differences between the $PM_{3.5}$ and $PM_{2.5}$ cutpoints and the PM_{11} and PM_{10} cutpoints are not likely (Willeke and Baron, 1993); thus, these results can be more readily compared. In what follows, the term "fine particles" refers to the $PM_{3.5}$ and $PM_{2.5}$ size fractions collected in the three studies.

Indoor-Outdoor Relationships. Outdoor concentrations of fine particles in five of the Harvard six cities and the two New York counties were relatively low, typically in the range of 10 to 20 $\mu g/m^3$ (Table 7-11). Only Steubenville, with an annual mean of 45 $\mu g/m^3$ (but a range among the outdoor sites of 20 to 60 $\mu g/m^3$) approached the mean outdoor level of 50 $\mu g/m^3$ observed in Riverside. It is interesting to note that average indoor concentrations

TABLE 7-11. INDOOR-OUTDOOR MEAN CONCENTRATIONS ($\mu g/m^3$) OF FINE PARTICLES IN THREE LARGE-SCALE STUDIES

Study Name	Homes	Out	In	In/Out
Harvard Six-City Study				
Portage, WI	11	10	20	2.0
Topeka, KS	10	10	22	2.2
Kingston-Harriman, TN	8	18	44	2.4
Watertown, MA	8	15	29	1.9
St. Louis, MO	10	18	42	2.3
Steubenville, OH	8	45	42	0.9
New York State ERDA Study				
Onondaga County	224	17	37	2.2
Suffolk County	209	22	46	2.1
EPA Particle TEAM Study				
Riverside, CA	178	50	43	0.9

Harvard: $PM_{3.5}$ measured using cyclone sampler. Samples collected every sixth day for one year (May 1986 to April 1987).

NYS: $PM_{2.5}$ measured using impactor developed at Harvard. Samples collected for one week at each household between January and April 1986.

PTEAM: $PM_{2.5}$ measured using Marple-Harvard-EPA sampler. Samples collected for two 12-h periods at each home between September and November 1990.

Source: Harvard data—Spengler et al. (1981); NYS data—Sheldon et al. (1989); PTEAM data—Pellizzari et al. (1992).

exceeded outdoor concentrations in the seven sites with low outdoor levels, (indoor/outdoor ratios were contained in a small range between 1.9 and 2.4), but were slightly less than outdoor concentrations in the two sites with high outdoor levels (ratios of 0.9).

Effect of Smoking. All three studies found cigarette smoking to be a major source of indoor fine particles. Multivariate calculations in all three studies result in rather similar estimates of the effect of smoking on fine particle concentrations. Spengler et al. (1981) estimated an increase of about 20 $\mu g/m^3$ per smoker based on 55 homes from all six cities. Since the 20 homes with at least one smoker averaged at least 1.25 smokers per home, this corresponds to about 25 $\mu g/m^3$ per smoking home. Spengler et al. (1985) found a smoking effect of about 32 $\mu g/m^3$ for smoking homes in multivariate models based on the Kingston-Harriman data. Santanam et al. (1990) found a smoking-related increase of 20-27 $\mu g/m^3$ in Steubenville and Portage (winter only) but only 10 $\mu g/m^3$ in Portage in summer. Sheldon et al. (1989) found an increase of 45 (Onondaga) and 47 (Suffolk) $\mu g/m^3$ per smoking home in a multivariate model of the New York State data. Özkaynak et al. (1993b) found an increase of about 27 to 32 $\mu g/m^3$ in

homes with smokers in a multivariate regression model of the PTEAM $PM_{2.5}$ data. Thus, the effect of a home with smokers on indoor fine particle concentrations was estimated to be about 20 to 30 $\mu g/m^3$ in the Six-City and PTEAM studies, but about 45 $\mu g/m^3$ in the New York State study.

Dockery and Spengler (1981a) found an effect of 0.88 $\mu g/m^3$ per cigarette for homes without air conditioning, and 1.23 $\mu g/m^3$ per cigarette for homes with air conditioning, based on 68 homes from all six cities. Lebret et al. (1987) found an effect of 0.8 $\mu g/m^3$ per cigarette for homes in the Watertown, MA, area. Leaderer and Hammond (1991) found an effect ranging between 1.9 and 2.3 $\mu g/m^3$ per cigarette contribution to a 24-h average. In a series of stepwise regressions on the PTEAM data, Özkaynak et al. (1993b) found an effect ranging between 1.2 and 2.4 $\mu g/m^3$ per cigarette smoked during a 24-h period. Taking the midpoint of these ranges leads to estimates for the Harvard Six-City, New York State and PTEAM studies of about 1.1, 2.1, and 1.8 $\mu g/m^3$ increases in fine particle concentrations per cigarette smoked in the home over a 24-h period.

Both the New York State study and the PTEAM study were able to estimate source strengths for different variables using a mass-balance model. The estimates for $PM_{2.5}$ emissions from cigarettes were very comparable, with Koutrakis et al. (1992) estimating 12.7 mg/cig compared to the PTEAM estimate of 13.8 mg/cig (Özkaynak et al., 1993a). Both studies also found similar elemental profiles for smoking, with potassium and chlorine being emitted in substantial amounts.

Effect of Other Variables. In the PTEAM Study, the second most powerful indoor source of PM_{10}, and possibly $PM_{2.5}$ particles, was cooking. Quite large emission strengths of several mg/minute of cooking were determined from the mass-balance model, while multiple regressions indicated that cooking could contribute between 10 and 20 $\mu g/m^3$ PM_{10}, and somewhat smaller amounts of $PM_{2.5}$, to the 12-h concentration.

Both the New York State and PTEAM studies also measured air exchange in every home, and both studies found that air exchange significantly affected indoor particle concentrations. In the PTEAM study, increased air exchange led to increased indoor air concentrations for both $PM_{2.5}$ and PM_{10} at night only, perhaps because outdoor concentrations were larger than indoor levels at night. In the New York State study, increased air exchange led to decreased RSP concentrations in Onondaga ($p < 0.02$) but no effect was noted in Suffolk ($p < 0.90$). In both of

these counties, indoor levels generally exceeded outdoor levels, so increased air exchange would generally reduce indoor concentrations.

7.2.2.2 Other Studies of PM Indoors

Several other large-scale studies of indoor PM in homes have taken place in other countries, and a number of smaller U.S. studies have been conducted. These are discussed below in order of the number of homes included in the study.

Lebret et al. (1990) carried out week-long RSP measurements (cutpoint not described) in 260 homes in Ede and Rotterdam, The Netherlands, during the winters of 1981 to 1982 and 1982 to 1983, respectively; 60% of the Ede homes and 66% of the Rotterdam homes included smokers. Diary information collected during the measurement period indicated that, on average, one to two cigarettes were smoked during the week, presumably by guests, even in the nonsmoking homes. Homes with one smoker averaged seven cigarettes smoked per day at home in Ede (N = 53) and 11 per day in Rotterdam (N = 35). Homes with two smokers averaged 21 cigarettes per day in Ede (N = 23) and 25 per day in Rotterdam (N = 15).

Geometric means for the combined smoking and nonsmoking homes were similar in the two cities (61 and 56 $\mu g/m^3$, respectively), with maxima of 560 and 362 $\mu g/m^3$. Outdoor concentrations averaged about 45 $\mu g/m^3$ (N not given). Indoor concentrations in the homes with smokers averaged about 70 $\mu g/m^3$ (calculated from data in the paper), compared to levels in the nonsmoking homes of about 30 $\mu g/m^3$. Multiple regression analysis indicated that the number of smoking occupants explained about 40% of the variation in the log-transformed RSP concentrations—family size, frequency of vacuuming, volume of the living room, type of space heating, and city (Ede versus Rotterdam) had no significant effect on RSP concentrations. In a second regression, the number of smoking occupants was replaced by the number of cigarettes and cigars smoked during the week. The regression equation was

$$\log(\text{RSP}) = 1.4 + 0.37 \log(\#\text{ cigarettes}) + 0.53 \log(\#\text{ cigars}) + 0.03 \log(\text{family size})$$

$$R^2 = 0.49;\ \text{d.f.} = 250\quad F = 83.7\quad p < 0.0001$$

From this equation, the authors estimated that one cigarette smoked per day would increase weekly average indoor RSP concentrations by 2 to 5 $\mu g/m^3$, whereas one cigar smoked per day

would increase indoor levels by 10 µg/m^3. Instantaneous RSP concentrations were measured using a TSI Piezobalance on the day the technicians were setting up the equipment. Table 7-12 shows the influence of smoking on these measurements.

TABLE 7-12. INFLUENCE OF RECENT CIGARETTE SMOKING ON INDOOR CONCENTRATIONS OF PARTICULATE MATTER[1]

Time Since Smoking	N	RSP (geom. mean) (μg/m^3)
No smoking	98	41
More than 1 h ago	18	52
Between 1/2 and 1 h ago	7	76
Less than 1/2 an hour ago	27	141
During the measurements	54	191

[1]Size cuts for measured particles not specified.

Source: Lebret et al. (1990).

Heavner et al. (1995) studied $PM_{3.5}$ at home and at work for 104 New Jersey and Pennsylvania females. The personal sampler used consisted of a cyclone sampling head attached to a 37-mm Fluoropore filter, connected by Tygon tubing to a 1.7 Lpm pump. The sampling head was worn on a lapel, collar, or pocket in the breathing zone of the participant until she went to bed, when the sampler was placed on the bedside table. The "home" pumps were turned on at 6 p.m. and sampled until about 8 a.m. the next morning (an average of 14 h); the "work" pumps were turned on at work and sampled for an average of 7 h. Participants were selected to include those with exposure to smoking at home or at work or both or neither. The 14-h evening and overnight concentrations in the homes averaged 86.7 ± 145.4 (SD) μg/m^3 for 30 homes with smokers and 27.6 ± 19.9 μg/m^3 for 58 homes without smokers. Corresponding values for workplaces were 67.0 ± 44.3 μg/m^3 for those 28 allowing smoking and 30.3 ± 17.6 μg/m^3 for the 52 without smoking, the differences being significant at $p < 0.0001$ (Wilcoxon rank sum) for both comparisons.

Diemel et al. (1981) measured particles in 101 residences in an epidemiological study related to a lead smelter in Arnhem, the Netherlands. The indoor sampler collected samples at a flowrate of 1 to 1.5 Lpm. The authors stated that particles ≤ 3 to 4 µm diameter should have

been sampled efficiently, but presented no data on measured cutpoint size. The outdoor samplers (number not given) were high-volume samplers. The 28-day average levels indoors ranged from 20 to 570 μg/m^3, with an arithmetic mean of 140 μg/m^3 (SD not presented) and a geometric mean of 120 μg/m^3; corresponding outdoor concentrations (2-mo averages of 24-h daily samples) ranged from 53.7 to 73.3 μg/m^3 (N not given), with nearly identical arithmetic and geometric means of 64 μg/m^3.

Kulmala et al. (1987) measured indoor and outdoor air in approximately 100 dwellings (including some office buildings) in Helsinki, Finland between 1983 and 1986. Samples were collected on Nuclepore filters using a stacked foil technique. The geometric mean for the combined fine particle (<1 μm) samples indoors was 16 μg/m^3, with a 95% range of 4 to 67 μg/m^3. The corresponding value for the indoor coarser particles (>1 μm) was 13 μg/m^3 with a range of 3 to 63 μg/m^3. Outdoors, the fine particles had a geometric mean of 20 μg/m^3 with a 95% range of 5 to 82 μg/m^3, and the coarser particles had a geometric mean of 16 μg/m^3 with a range of 3 to 91 μg/m^3.

Quackenboss et al. (1989) reported PM_{10} and $PM_{2.5}$ results from 98 homes in the Tucson, AZ area selected as part of a nested design for an epidemiological study. The Harvard-designed dual-nozzle indoor air sampler (Marple et al., 1987) was used for indoor air measurements. Outdoor air was measured within each geographic cluster by the same instrument; supplementary data were obtained from the Pima County Air Quality Control District, but these data did not include $PM_{2.5}$ measurements and some data were apparently PM_{15}. Homes were classified by (a) tobacco smoking and (b) use of evaporative ("swamp") coolers, which apparently act as a removal mechanism for particles (Table 7-13). Homes without smoking averaged about 15 μg/m^3 $PM_{2.5}$, compared to 27 μg/m^3 for homes reporting one or less pack a day, and 61 μg/m^3 for homes reporting more than one pack a day. $PM_{2.5}$ particles accounted for about half of the PM_{10} fraction in nonsmoking homes, increasing with the amount of smoking to about 80% in those homes with heavy smoking. Outdoor PM_{10} particles were not strongly correlated with indoor levels ($R^2 = 0.18$; $N \approx 90$).

TABLE 7-13. INDOOR AVERAGE $PM_{2.5}$ AND PM_{10} ($\mu g/m^3$) BY REPORTED SMOKING IN THE HOME AND EVAPORATIVE COOLER USE DURING SAMPLING WEEK FOR TUCSON, AZ STUDY

Smoking Cigarettes/Day	Evaporative Cooler	$PM_{2.5}$			PM_{10}		
		Mean	S.D.	Homes	Mean	S.D.	Homes
None	Yes	8.8	5.0	(20)	21.0	9.7	(20)
	No	20.3	19.0	(25)	38.4	22.9	(23)
	Total	15.2	15.5	(45)	30.3	19.9	(43)
1-20	Yes	19.3	8.8	(10)	33.9	12.0	(10)
	No	32.3	28.5	(16)	53.4	33.9	(17)
	Total	27.3	23.6	(26)	46.2	29.1	(27)
>20	Yes	36.2	32.9	(8)	47.4	39.6	(9)
	No	82.7	55.4	(9)	102.5	60.6	(9)
	Total	60.8	50.8	(17)	75.0	57.2	(18)

$PM_{2.5}$: Significant ($p < 0.01$) main effects for smoking and evaporative cooler use; two-way interaction nearly significant ($p = 0.06$).
PM_{10}: Significant ($p < 0.01$) main effects for evaporative cooler and smoking.

Source: Quackenboss et al. (1989).

Quackenboss et al. (1991) extended the analysis of the Tucson homes over three seasons. Median indoor $PM_{2.5}$ levels in homes with smokers were about 20 $\mu g/m^3$ in the summer and spring/fall seasons compared to about 10 $\mu g/m^3$ in homes without smokers in those seasons. In winter, however, the difference was considerably increased, with the median level in 24 homes with smokers at about 36 $\mu g/m^3$ compared to 13 $\mu g/m^3$ in 26 homes without smokers.

Sexton et al. (1984) reported on a study in Waterbury, VT. This study included 24 homes, 19 with wood-burning appliances, and none with smokers. 24-h samples were collected in each home every other day for two weeks, providing 163 valid indoor samples. Indoor RSP levels ranged from 6 to 69 $\mu g/m^3$ with a mean value of 25 $\mu g/m^3$. Outdoor levels ranged from 6 to 30 $\mu g/m^3$ with a mean value of 19 $\mu g/m^3$. Indoor concentrations were not significantly correlated with outdoor concentrations ($r = 0.11$, $p > 0.16$.)

Kim and Stock (1986) reported results for 11 homes in the Houston, TX area. (Year and the season not reported in the paper.) For most homes, two 12-h $PM_{2.5}$ samples (day and night) were collected for approximately one week. Sampling methods were not fully discussed, but apparently they involved samples collected using a mobile van near each home. The mean weekly concentrations in the five smoking homes averaged 33.0 ± 4.7 (SD) $\mu g/m^3$, versus mean outdoor concentrations averaging 24.7 ± 7.4 $\mu g/m^3$ (calculated from data presented in paper).

Indoor concentrations in the six nonsmoking homes averaged 10.8 ± 4.9 μg/m^3 compared to outdoor levels of 12.0 ± 5.9 μg/m^3.

Morandi et al. (1986) reported on 13 Houston, TX, homes monitored during 1981 as part of a larger personal monitoring study of 30 nonsmoking participants. The TSI Piezobalance (cutpoint at about $PM_{3.5}$) was employed for personal monitoring, with technicians "shadowing" the participants and taking consecutive 5-min readings. At the homes, dichotomous samplers (cutpoints at $PM_{2.5}$ and PM_{10}) were used for two 12-h daytime samples (7 a.m. to 7 p.m.) both inside and outside the homes for seven consecutive days. Little difference was noted in the indoor concentrations at homes (25 ± 30 (SD) μg/m^3) and at work or school (29 ± 25 μg/m^3). The highest overall respirable suspended particle (RSP) concentrations occurred in the presence of active smoking (89 μg/m^3), significantly different from mean RSP values measured in the absence of smokers (19 μg/m^3; $p < 0.0001$). Among homes with smokers, those homes with central air conditioning were significantly ($p<0.0001$) higher (114 versus 52 μg/m^3) than those with no air conditioning. Cooking was associated with significantly higher RSP concentrations (27 μg/m^3 compared to 20 μg/m^3, $p < 0.01$). The single highest RSP concentration (202 μg/m^3) was found in a home with no smokers and no air conditioning but with active cooking. The authors concluded that cooking was a more important source of indoor RSP than smoking, at least in the few homes they studied.

Coultas et al. (1990) measured $PM_{2.5}$ in 10 homes containing at least one smoker, using the Harvard aerosol impactor. Samples were collected for 24 h every other day for 10 days and then for 24 h every other week for 10 weeks, resulting in 10 samples per household. The mean concentrations of $PM_{2.5}$ ranged from 32.4 ± 13.1 (SD) to 76.9 ± 32.9 μg/m^3. Outdoor particle concentrations were not reported; thus it is difficult to calculate the portion of the observed $PM_{2.5}$ that might be due to ETS.

Kamens et al. (1991) measured indoor particles in three homes without smokers in North Carolina in November and December 1987 (no measurements of outdoor particles were taken). Two dichotomous samplers ($PM_{2.5}$ and PM_{10}), several prototype personal samplers (also $PM_{2.5}$ and PM_{10}), three particle sizing instruments including a TSI electrical aerosol mobility analyzer (EAA) with 10 size intervals between 0.01 and 1.0 μm, and two optical scattering devices covering the range of 0.09 to 3.0 and 2.6 to 19.4 μm were employed. Air exchange measurements were made using SF_6 decay over the course of the seven 8-h (daytime) sampling

periods. There were also three 13-h (evening and overnight) sampling periods. For the entire study, 37% of the estimated total mass collected was in the fine fraction, and another 37% was > 10 μm. The remainder (26%) was in the inhalable coarse (PM_{10} – $PM_{2.5}$) fraction. However, considerable variation was noted in these size distributions. For example, on one day with extensive vacuuming, cooking, and vigorous exercising of household pets, 52% of the total mass appeared in the fraction >10 μm, but only 18% in the fine fraction. The peak in particle mass on that day coincided with vacuuming and sweeping of carpets and floors. On another day, cooking of stir-fried vegetables and rice produced a large number of small particles, with those <0.1 μm accounting for 30% of the total EAA particle volume, much more than the normal amount. The cooking contribution of that one meal to total 8-h daytime particle volume exposure was calculated to be in the range of 5 to 18%. The authors concluded that the most significant indoor source of small particles (<2.5 μm) in all three of these nonsmoking homes was cooking, while the most significant source of large particles (>10 μm) was vacuum sweeping. Inhalable coarse particles (PM_{10} – $PM_{2.5}$) appeared to be of largely biological (human dander and insect parts) and mineral (clay, salt, chalk, etc.) origin.

In a test of a new sampling device (a portable nephelometer), Anuszewski et al. (1992) reported results from indoor and outdoor sampling at nine Seattle, WA, homes sampled for an average of 18 days each during the winter of 1991 to 1992. The nephelometer is a light-scattering device with rapid (1-min) response to various household activities such as sweeping, cigarette smoking, frying, barbecuing, and operating a fireplace. Homes with fewer activities showed high correlations of indoor and outdoor light-scattering coefficients, both between hourly averages and 12-h averages. However, homes with electrostatic precipitators, with weather-stripped windows or doors, and with gas cooking or heating devices showed weak 12-h indoor-outdoor correlations.

Chan et al. (1995) studied particles and nicotine in seven homes with one smoker each in Taiwan. Sampling was carried out in summer and winter of 1991. Each home had one indoor PM_5 sampler in the living room and another in the yard. In the winter study, two homes had PM_{10} samplers added inside and outside and at two central sites. Indoor mean PM_5 concentrations averaged 44 ± 32 (SD) $\mu g/m^3$ in summer compared to outdoor levels of 27 ± 15 $\mu g/m^3$. Corresponding winter values were 107 ± 44 $\mu g/m^3$ and 92 ± 40 $\mu g/m^3$.

Daisey et al. (1987) measured RSP, PAH, and extractable organic matter (EOM) in seven Wisconsin homes with wood stoves; one 48h (1,000 m^3) sample was collected during woodburning and a second sample was collected when no woodburning occurred. Five of seven homes had somewhat higher RSP levels during woodburning, but the mean difference was not significant.

Highsmith et al. (1991) reported on two 20-home studies in Boise, ID and Roanoke, VA. The Boise study assessed the effects of wood burning on ambient and indoor concentrations in the area. Ten homes with wood burning stoves were matched with 10 homes without such stoves. One matched pair of homes was monitored from Saturday through Tuesday for eight consecutive 12-h periods. Ambient $PM_{2.5}$ levels increased by about 50% at night, suggesting an influence of woodburning. Indoor $PM_{2.5}$ concentrations also were increased (by about 45%) in the homes with the wood burning stoves compared to those without (26.3 versus 18.2 $\mu g/m^3$), although coarse particles showed no increase (10.2 versus 9.7 $\mu g/m^3$). The Roanoke study, designed to assess the effects of residential oil heating, showed no effects on indoor levels of fine or coarse particles.

Löfroth et al. (1991) measured particle emissions from cigarettes, incense sticks, "mosquito coils," and frying of various foods. Emissions were 27 and 37 mg/g for two brands of Swedish cigarettes, 51 and 52 mg/g for incense sticks and cones, and 61 mg/g for the mosquito coil. Emissions from frying pork, hamburgers, herring, pudding, and Swedish pancakes ranged from 0.07 to 3.5 mg/g.

Mumford et al. (1991) measured PM_{10}, PAH, and mutagenicity in eight mobile homes with kerosene heaters. Each home was monitored for 2.6 to 9.5 h/day (mean of 6.5 h) for three days a week for two weeks with the kerosene heaters off and for two weeks with them on (average on-time of 4.5 h). Mean PM_{10} levels were not significantly increased when the heaters were on (73.7 ± 7.3 (SE) $\mu g/m^3$ versus 56.1 ± 5.7 $\mu g/m^3$), but in two homes levels increased to 112 and 113 $\mu g/m^3$ when the heaters were on. Outdoor concentrations averaged 18.0 ± 2.1 $\mu g/m^3$.

Colome et al. (1990) measured particles using PM_{10} and PM_5 (cyclone) samplers inside and outside homes of 10 nonsmokers, including eight asthmatics, living in Orange County, CA. Indoor PM_{10} samples were well below outdoor levels for all homes (mean of 42.5 ± 3.7 (SE) $\mu g/m^3$ indoors versus 60.8 ± 4.7 $\mu g/m^3$ outdoors). No pets, wood stoves, fireplaces, or kerosene heaters were present in any of these homes.

Lioy et al. (1990) measured PM_{10} at eight homes (no smokers) for 14 days in the winter of 1988 in Phillipsburg, NJ, which has a major point source consisting of a grey-iron pipe manufacturing company. The Harvard impactor was used indoors to collect 14 24-h samples beginning at 4:30 p.m. each day; Wedding hi-vol PM_{10} samples were deployed at three outdoor sites. A fourth outdoor site was located on a porch of one of the homes, directly across the street from the pipe manufacturer. The first three sites showed little difference from one another, whereas on day 4 and day 6 of the study, the outdoor sampler on the porch had readings that were considerably (about 40 $\mu g/m^3$) higher than the other outdoor samplers, suggesting an influence of the nearby point source. The geometric mean outdoor PM_{10} concentration was 48 $\mu g/m^3$ (GSD not provided) compared to 42 $\mu g/m^3$ indoors. A simple regression equation for all homes (N = 101 samples) explained 45% of the cross-sectional variance in indoor PM_{10}:

$$\text{Indoor } PM_{10} = 0.496 \text{ Outdoor } PM_{10} + 21.5 \quad (R^2 = 0.45)$$

However, individual regressions by home showed much better R^2 values in most cases, ranging from 0.36 to 0.96 (Table 7-14). All slopes were significant.

Thatcher and Layton (1995) measured optical particle size distributions inside and outside a residence in the summer. Measured deposition velocities for particles between 1 and 5 µm closely matched the calculated gravitational settling velocities; however, for particles >5 µm, the deposition velocity was less than the calculated settling velocity, perhaps due to the non-spherical nature of these particles. The deposition velocities determined by the authors corresponded to a particle deposition rate *k* of 0.46 h^{-1} for particles of size range 1 to 5 µm and 1.36 h^{-1} for particles of size range 5 to 10 µm. These values are very comparable with the values of 0.39 h^{-1} for particles less than 2.5 µm and 1.01 h^{-1} for particles between 2.5 and 10 µm found by the PTEAM Study. The authors measured the penetration factor *P* by the following method: They first carried out vigorous house cleaning activities to raise the level of resuspended dust well above outdoor levels.

TABLE 7-14. REGRESSION OF INDOOR ON OUTDOOR PM_{10} CONCENTRATIONS: THEES STUDY, PHILLIPSBURG, NJ ($\mu g/m^3$)

House	N	Intercept	SE	p	Slope	SE	p	R^2
1	14	19	9	NS	0.44	0.06	S	0.79
2	14	16	14	NS	0.40	0.08	S	0.68
3	9	9	5	NS	0.55	0.04	S	0.96
4	14	20	21	NS	0.73	0.15	S	0.66
5	13	6	10	NS	0.43	0.07	S	0.75
6	13	−1	18	NS	0.89	0.13	S	0.81
7	12	24	25	NS	0.70	0.29	S	0.36
8	14	27	8	S	0.54	0.05	S	0.91

S = Significant
NS = Non-significant

Source: Data from THEES Study (Lioy et al., 1990).

They then left the house, while automated instruments measured the deposition rate *k* for the different particle sizes and the air exchange rate *a* for SF_6 tracer gas. With these values of *a* and *k* in hand, they solved the equation for *P*, using the steady-state values for C_{in} and C_{out} observed long after the dust had settled:

$$P = \frac{C_{in}\ (a+k)}{C_{out}\ a} \tag{7-6}$$

For all size ranges tested, including the largest (10 to 25 μm), the experimentally determined value for *P* was not significantly different from 1 (Figure 7-13). This result is in agreement with the PTEAM conclusion that *P* is 1 for both fine and coarse particles, although the latter conclusion was derived from a nonlinear (statistical) approach whereas the present result was experimentally obtained.

The resuspension results of Thatcher and Layton (1995) (Figure 7-14) show the effect of a vigorous housecleaning activity. The authors concluded "Although particles larger than 5 μm show significant resuspension in these experiments, particles smaller than 5 μm are not readily resuspended, and particles less than 1 μm show almost no resuspension even with vigorous activity." Figure 7-15 shows that just one person walking in and out of a carpeted

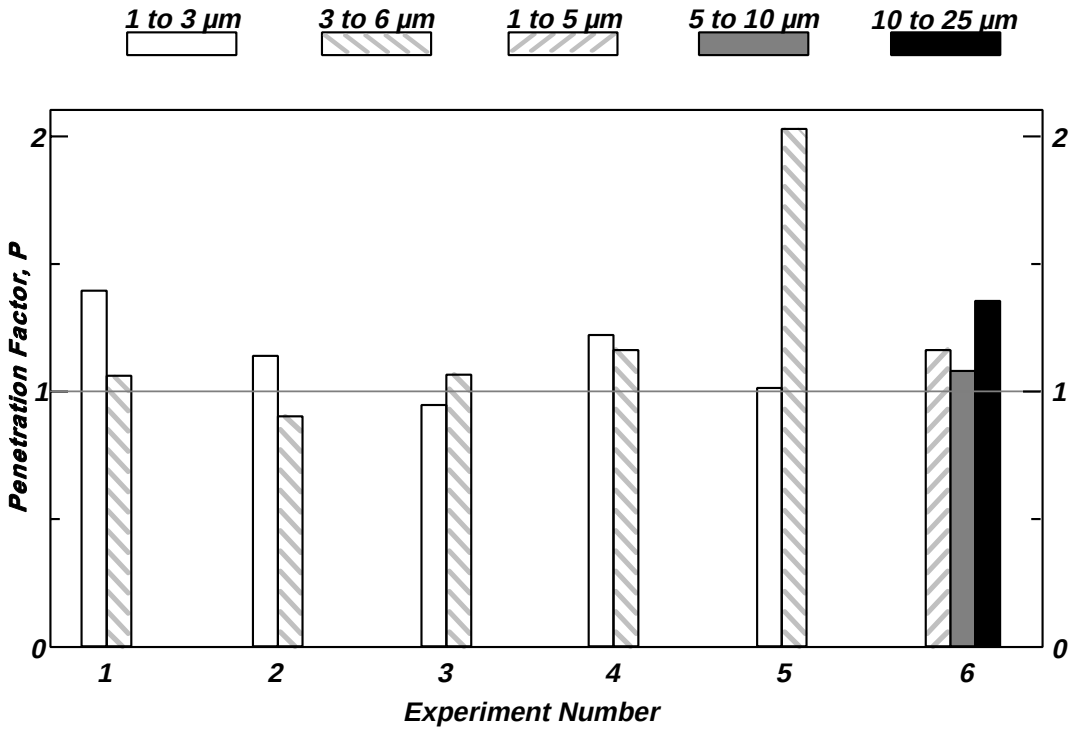


Figure 7-13. Results of six penetration experiments in a test home.

Source: Thatcher and Layton (1995).

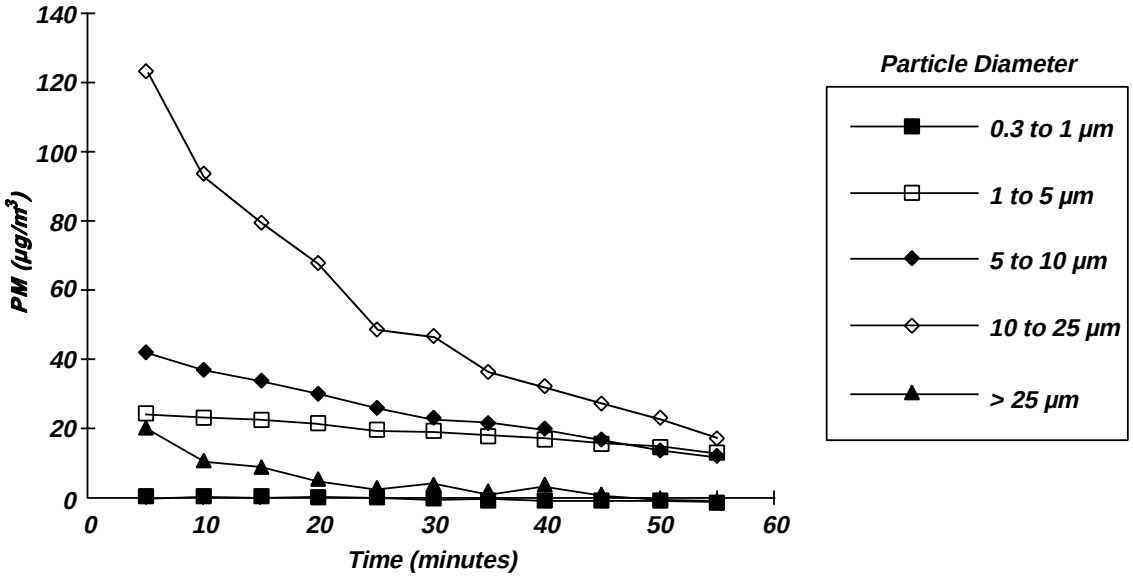


Figure 7-14. The change in suspended particle mass concentration versus time, as measured by optical particle counter, assuming spherical particles of unit density. All resuspension activities are stopped at t = 0.

Source: Thatcher and Layton (1995).

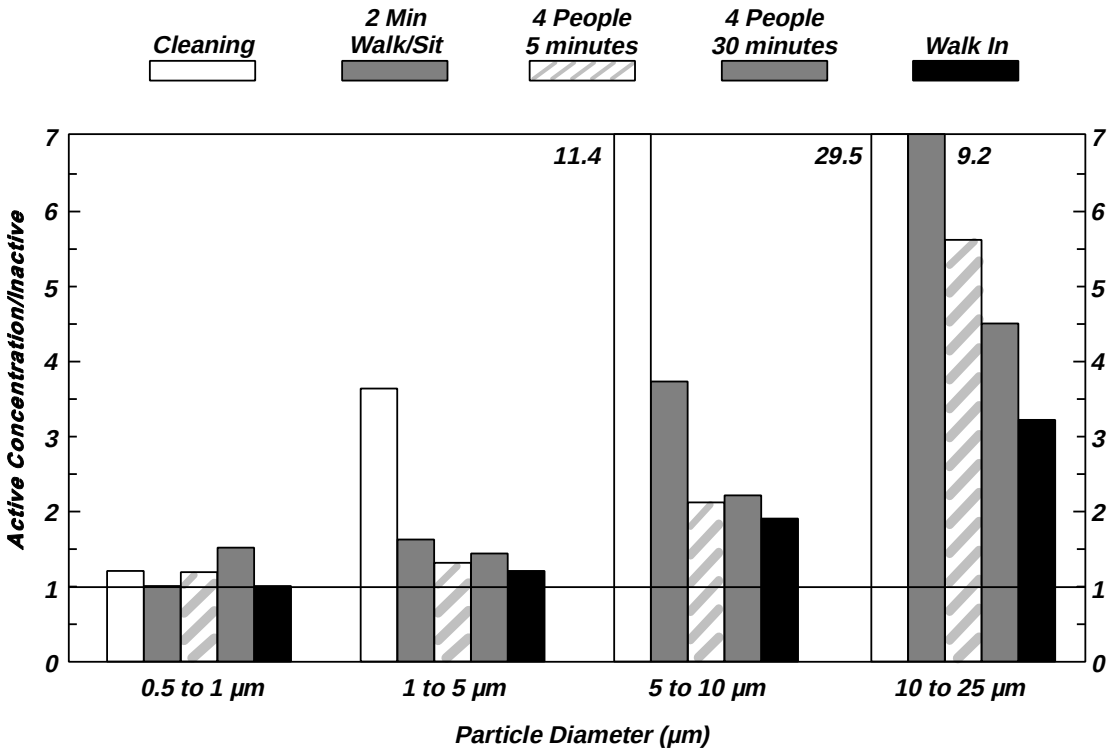


Figure 7-15. The ratio of the suspended particle concentration after a resuspension activity to the indoor concentration before that activity, by particle size. The activities tested are (1) vigorous vacuuming and housecleaning, (2) 2 min of continuous walking and sitting in the living area by one person, (3) 5 min of normal activity by four people, (4) 30 min of normal activity and (5) one person walking into and out of the living area.

Source: Thatcher and Layton (1995).

living area can increase indoor particle concentrations in the ranges 5 to 10 µm by 100% and 10 to 25 µm by 200%. The absolute increase in indoor concentrations by this activity is a function of the surface dust loading in those size ranges. Surface dust loadings ($\mu g/m^2$) increase with the time since last cleaning (Raunemaa et al., 1989; Wilmoth et al., 1991).

Because fluffy house dust can be resuspended, it will contribute to total airborne exposure to particles and constituents such as metals and pesticides. Roberts et al. (1990) studied 42 homes in Washington State. Geometric mean lead concentration in 6 homes where shoes were removed on entry was 240 $\mu g/m^2$ on carpets, compared to 2,900 $\mu g/m^2$ on carpets in 36 homes where shoes were kept on. In Japan, where shoes are removed on entry and straw mats (tatami) are usually used instead of carpets, Tamura et al. (1996) found evidence of negligible

PM_{10} resuspension. These findings suggest that most of the carpet dust in a home enters via track-in on shoes rather than by infiltration of ambient air.

7.2.2.3 Personal Exposures to Environmental Tobacco Smoke.

Jenkins et al. (1995a) reported on the first 12 cities of a 16-city sampling survey comparing ETS exposures at home and at work. About 100 nonsmoking persons in each city were recruited to wear a personal monitor at work and another personal monitor away from work. The monitors collected $PM_{3.5}$ particles, which were then analyzed for tobacco smoke markers (UVPM, FPM and solanesol). Nicotine and other gas-phase markers were also collected. Subjects provided saliva samples, which were used to screen out smokers reporting themselves as nonsmokers. (Using different cutoff points of 10, 30, or 100 µg/L, between 1.82 and 5.2% of the 1073 subjects would have been misclassified as nonsmokers). Four cells were defined: persons exposed to smoke at home and at work (N = 119); persons exposed at home but not at work (110); persons exposed at work but not at home (163); and persons exposed neither at home nor at work (504). All four particle markers agreed in ranking the four cells for total ETS exposure in the order listed—that is, nonwork (including home) ETS exposures were greater than work exposures as shown in Table 7-15. The authors identified several problems with the selection of the sample. First, the sample was 68% female. Secondly, the socioeconomic level was biased high, with about twice as many persons having some college or being college graduates as the population as a whole. It is well known that smoking rates decrease as education and income rise, and this study confirmed that observation--when broken out by income, ETS markers decreased by factors of 2 to 5 as annual income rose from $10,000 to $100,000. The authors compared ETS levels in offices with no smoking (N = 629), restricted smoking (N = 297) and unrestricted smoking (N = 113). Median (mean) levels of RSP increased from 13 (18) to 16 (28) to 33 (58) µg/m^3 in the three categories, with corresponding nicotine medians (means) of 0.025 (0.11), 0.09 (0.87), and 0.44 (2.7) µg/m^3.

Jenkins et al. (1995b) updated the results to the full 16 cities. The final number of participants in the four cells were 157, 234, 281, and 808, respectively. The median RSP ($PM_{3.5}$) values changed only slightly, increasing to 33.6 from 32 μg/m^3 in Cell 1 and decreasing to 23.3 μg/m^3 in Cell 2, with no changes in the remaining two cells.

TABLE 7-15. MEDIAN VALUES (μg/m^3) FOR ENVIRONMENTAL TOBACCO SMOKE MARKERS

Cell (N)	Nonwork	Work	RSP	UVPM	FPM	Solanesol	Nicotine
1 (119)	S	S	32	12	7.7	0.113	1.46
2 (110)	S	NS	24	7.6	5.9	0.058	0.56
3 (163)	NS	S	20	2.3	1.2	0.003	0.11
4 (504)	NS	NS	15	1.1	0.6	ND	0.02

S = smoker; NS = nonsmoker; ND = not detectable.

Source: Jenkins et al. (1995a).

ETS Exposures in Restaurants and Buildings. Oldaker et al. (1993) reported results of analyzing ETS markers in four office buildings. Median RSP levels were 30 and 34 μg/m^3 in two buildings allowing smoking, compared to 5 and 7 μg/m^3 in two buildings without smoking. Crouse et al. (1989) reported on measurements of RSP ($PM_{3.5}$) in 42 North Carolina restaurants. Geometric mean (arithmetic mean) values were 5.3 (8.6), 26.1 (34.1) and 62.0 (80.8) μg/m^3, respectively. Oldaker et al. (1990) measured $PM_{3.5}$ in 33 restaurants in the Winston-Salem, NC, area during the summer of 1986 and the winter of 1988 to 1989; in the winter, the cutpoint was changed to $PM_{2.5}$. A wide range of particle concentrations was noted, from 18 to 1,374 μg/m^3 in the summer, and <25 to 281 μg/m^3 in winter.

7.2.2.4 The Fraction of Outdoor Air Particles Penetrating Indoors

Having reviewed the literature on particles in homes, it is useful to return to one of the questions we asked at the outset: For a home with no indoor sources or resuspension of settled dust of ambient origin, how much protection is offered against outdoor particles of various size ranges?

The governing equation in this case is

$$\frac{C_{in}}{C_{out}} = \frac{Pa}{a+k} \qquad (7\text{-}6)$$

Thus, there are three parameters affecting the fraction of outdoor air particles to be found indoors: the penetration factor *P*, the air exchange rate *a*, and the particle deposition rate *k*.

Penetration Factor P. The penetration factor *P* is a measure of the ability of a gas or particle to penetrate the building envelope; $0 \leq P \leq 1$. For a nonreactive gas, such as CO, the factor is expected to be 1. For large particles, the factor would be expected to go to zero with increasing particle size and decreasing air exchange rate. The question is at what combinations of size range and air exchange rate does the factor *P* begin to decrease significantly from unity for PM?

Two recent studies have attempted to determine the value of *P* for different particle size ranges. The PTEAM study (Özkaynak et al., 1996) found a value of $P \approx 1$ for both $PM_{2.5}$ and PM_{10} particles. The value was determined statistically by a nonlinear solution of Equation 7-5 (including all indoor sources) for 178 homes. Thatcher and Layton (1995) also found a value of $P \approx 1$ for all size ranges tested, including the ranges 1 to 3 μm, 3 to 6 μm, 1 to 5 μm, 5 to 10 μm, and 10 to 25 μm. The authors determined their values experimentally by direct measurement on one instrumented house. The results for the first two size ranges were obtained in five replicate experiments; for the last three size ranges, in only one experiment (Figure 7-13). Thus the two studies used different methods but arrived at the same conclusion: *particles less than 10 μm in aerodynamic diameter penetrate building envelopes with an efficiency approaching that of nonreactive gases.* Clearly, more work needs to be done to test this finding at lower air exchange rates.

Air Exchange Rate a. Air exchange rates in residences depend on three major factors: building construction, ambient conditions, and resident activities.

The building construction determines the lower bound of the air exchange rate. That is, rates cannot be reduced below the rate allowed by diffusion through the building cracks, holes, and other uncontrolled means of particle ingress in the absence of wind and buoyancy differences. Tests by building pressurization (e.g., using "blower doors") are able to determine a parameter ("crack length") that quantifies this lower bound. Buildings that are extremely tightly constructed for energy efficiency are able to reduce the lower bound of the air exchange rate to the order of 0.1 air change per hour (ach, or h^{-1}).

Ambient conditions, particularly temperature and wind velocity, can also drive air exchange rates. Strictly speaking, it is the *difference* between indoor and outdoor temperatures that creates either a pressure difference (closed windows) or a convective behavior (open windows) leading to higher air exchange rates as the temperature difference increases. As wind velocity rises, pressure differences also increase and therefore the air exchange rate rises. Besides these immediate ambient conditions we also have climatic conditions. A region that can expect a daily sea breeze is more likely to use open windows than air conditioning for ventilation. Northern areas are more likely to have tightly constructed buildings than southern areas in the USA.

In most cases, by far the most important factor affecting air exchange rates is the behavior of the resident(s). This includes such considerations as the number of residents, the number and age of children, the number of pets that spend time outdoors, whether or not air conditioning is used, and how much time doors and windows are open. Since residents are more active during the day, and doors are opened and closed more often, air exchange rates during the day typically exceed those at night, both in winter and in summer. In the PTEAM Study, the median daytime air exchange rate was 1.02 h^{-1} compared to an overnight median of 0.80 h^{-1} (Wallace et al., 1993). In the Parkville community of Baltimore, MD, in the spring, the daytime median was 0.40 h^{-1} and the overnight median was 0.28 h^{-1}. In Los Angeles coastal communities in the summer, the daytime median was 2.2 h^{-1} and the overnight median was 1.2 h^{-1}. (All values derived from U.S. Environmental Protection Agency, 1995)

Fortunately, a large number of surveys have been carried out in which air exchange rates of homes have been measured. These include the three major particle studies already mentioned, and some studies of other pollutants. A paper collecting results from many surveys found a geometric mean for 2844 U.S. residences of 0.53 h^{-1} with a geometric standard deviation of 2.3 (Murray and Burmaster, 1995). The mean value for all 2844 homes was 0.76 h^{-1}, which corresponded to the 70th percentile. However, the geometric means varied by season (a low of 0.31 h^{-1} in fall and a high of 1.00 h^{-1} in summer) and by region (a low of 0.31 h^{-1} in the North and a high of 0.69 h^{-1} in the South—mainly southern California). The geometric standard deviations for individual seasons and regions were generally very close to 2, ranging from 1.9 to 2.5. (It should be noted here that the homes were not selected to represent the nation, and that there are very great disparities in the number of homes sampled in any one region.)

A second paper (Koontz and Rector, 1995) used a nearly identical data set, but weighted the 2889 measured homes by the state populations to estimate more closely the national distribution. Their estimates are similar to those of Murray and Burmaster (1995) with an arithmetic mean of 0.63 h^{-1}, a geometric mean of 0.46 h^{-1} and a GSD of 2.25.

However, certain smaller areas with pronounced climatic conditions could have very much higher air exchange rates. In a region such as the South Bay of Los Angeles, Wallace et al. (1991c) found that 49 of 50 homes had no air conditioning and depended on the daily land-sea breeze for ventilation. In this area, winter air exchange rates had a geometric mean of 0.75 h^{-1} and summer air exchange rates were much higher, with a geometric mean of 2.16 h^{-1}. Both these ranges are much higher than the typical values reported above. Thus, it is important to consider the individual geographic region of study and its local climatic characteristics before selecting a range of air exchange rates to characterize the region.

With that caveat, the empirical distribution for a large number of U.S. homes across all seasons, but with disparate representation among the various regions of the country, appears to have a median value of about 0.5 h^{-1}, with a one geometric standard deviation ($\pm \sigma$) range of 0.2 to 1.1 h^{-1}, and a $\pm 2\sigma$ range of 0.1 to 2.2 h^{-1} (Murray and Burmaster, 1995; Koontz and Rector, 1995).

Deposition Rate k. In a residence, the deposition rate *k* depends on many factors, such as scale of turbulence, and the size, shape, electrostatic charge, and density of the particle. For larger particles, the deposition rate is determined largely by gravitational settling; for smaller particles, deposition on vertical surfaces by diffusion may also be important (Nazaroff et al., 1993). Unfortunately, fine particle deposition rates are not well characterized. Typically, one must measure over very long periods of time (weeks to months) to collect enough particles for analysis by sophisticated techniques. A series of studies in nearly unoccupied buildings containing telephone-switching electrical equipment resulted in average values for the deposition velocity of sulfate particles ranging from 0.003 to 0.005 cm/s (Sinclair et al., 1988, 1990, 1992; Weschler et al., 1989); these values correspond to values of *k* (using a surface to volume ratio of 3 m^{-1}) of 0.3 to 0.5 h^{-1}. However, another series of studies in museums resulted in values an order of magnitude smaller (Ligocki et al., 1990; Nazaroff et al., 1990a,b). Results for the sulfur ($PM_{2.5}$) deposition rate in the PTEAM studies were 0.16 h^{-1}, lying between the values found by

these two groups. Nazaroff et al. (1993) concluded that deposition rates could vary as a result of different surfaces or near-surface air flows, amount of thermal isolation of the surfaces from building walls, turbulence, and many other factors. Thus it is not likely that theoretical calculations of deposition rates will provide trustworthy estimates. Nor is it likely that chamber studies, with their limited ability to reproduce the variety of floor coverings and air flows found in residences, can provide much information relevant to real-world residences.

In the absence of precise theory or widely applicable chamber study estimates, the largest study of residences including a calculation of empirical deposition rates is the PTEAM study. The estimate for $PM_{2.5}$ was 0.39 h^{-1}, for PM_{10} it was 0.65 h^{-1}, while for the coarse fraction (the difference between PM_{10} and $PM_{2.5}$) it was 1.01 h^{-1}.

What Is the Fraction of Outdoor Air Particles Found Indoors at Equilibrium?

Based on the values of *P*, *a*, and *k* discussed above, an answer can be provided to this question. Figure 7-16 shows the fraction of outdoor fine and coarse particles found in homes under equilibrium conditions for a range of air exchange rates. This fraction is calculated using the value of $P = 1$ determined in the PTEAM and the Thatcher and Layton (1995) studies, and the values of *k* for fine and coarse particles calculated in the PTEAM study. The fractions are displayed over the 95% range of observed air exchange rates (0.1 to 2.2 h^{-1}) in studies reported on by Murray and Burmaster (1995). It can be seen that at the mean air exchange rate of 0.76 h^{-1} reported in Murray and Burmaster (1995), the fractions of outdoor fine (<2.5 μm) and coarse particles (>2.5 and <10 μm) that will be found indoors under equilibrium conditions are 66% and 43%, respectively. The fraction of PM_{10} found indoors will lie between these two curves, with the exact placement dependent on the relative proportions of fine and coarse particles constituting the PM_{10}.

The actual distribution of values of $a/(a+k)$ observed in the PTEAM Study is provided in Table 7-16 for PM_{10} and for its fine and coarse fractions. As can be seen, the average values across day and night were about 67% for fine particles and 47% for coarse particles, with PM_{10} exactly between the two size fractions at 57%.

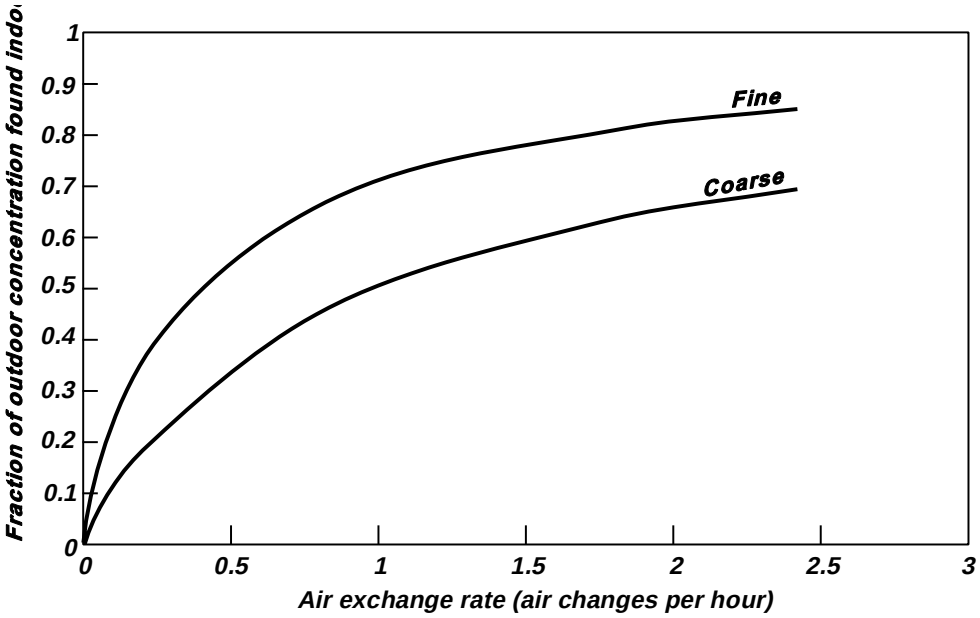


Figure 7-16. Fraction of indoor particulate matter (PM) from outdoor airborne PM, under equilibrium conditions, as a function of air-exchange rate, for two different size fractions.

Source: Calculated from PTEAM database (Özkaynak et al., 1993a; Wallace, 1996).

These results suggest that if persons at risk of health effects from outdoor particle pollution are able to significantly decrease the air exchange rates in their homes (by weatherization, installation of air conditioning to reduce use of windows, etc.) they could decrease the fraction of outdoor air particle concentration in their homes. A decrease in the air exchange rate from the mean level of 0.76 h^{-1} reported above to an achievable (16th percentile) value of 0.25 h^{-1} would decrease the indoor air level of outdoor-generated fine $PM_{2.5}$ particles from 66% to 39% of the outdoor level, and of PM_{10} from 54% to 28%.

7.2.2.5 Studies of PM in Buildings

The single largest study of particles in buildings was carried out by the Lawrence Berkeley Laboratory (LBL) for the Bonneville Power Administration (BPA) (Turk et al., 1987, 1989). Thirty-eight buildings were chosen from two climatic regions in the Pacific Northwest: Portland-Salem, OR (representing mild coastal conditions), and Spokane-Cheney,

TABLE 7-16. FRACTION OF CONCENTRATION OF OUTDOOR PARTICLES ESTIMATED TO BE FOUND INDOORS AT EQUILIBRIUM: RESULTS FROM THE PARTICULATE TOTAL EXPOSURE ASSESSMENT METHODOLOGY STUDY

	Daytime (N=174)			Overnight (N=175)		
Statistic	Fine	PM_{10}	Coarse	Fine	PM_{10}	Coarse
Mean	0.68	0.58	0.49	0.66	0.55	0.46
Standard deviation	0.17	0.19	0.20	0.15	0.17	0.17
Standard error	0.013	0.015	0.015	0.012	0.013	0.013
Geometric mean	0.66	0.55	0.45	0.64	0.53	0.42
Minimum	0.28	0.19	0.13	0.28	0.19	0.13
25th percentile	0.55	0.42	0.32	0.55	0.43	0.32
Median	0.70	0.58	0.47	0.66	0.54	0.43
75th percentile	0.83	0.75	0.65	0.79	0.69	0.59
Maximum	0.95	0.93	0.89	0.94	0.90	0.85

Fractions calculated from the formula $Pa/(a+k)$, where
$P = 1$;
$k = 0.39\ h^{-1}$ for fine particles, $PM_{2.5}$;
$k = 0.65\ h^{-1}$ for PM_{10}; and
$k = 1.01\ h^{-1}$ for coarse particles $2.5\ \mu m < AD < 10\ \mu m$.
Values for a measured in 175 homes during the PTEAM Study.

Source of data: Values calculated from PTEAM database (Wallace, 1996).

WA (representing extreme inland conditions). The buildings were studied for a variety of pollutants to determine how ventilation rates affect indoor air quality. Buildings were measured in winter (21 buildings in both regions), spring (10 buildings in both regions) and summer (nine buildings in the inland region only). All but four buildings were government or public properties, and therefore the 38 buildings cannot be considered to represent the full mix of building types.

Each building was monitored for 10 working days over a two-week period. From four to eight particle sampling sites were chosen in each building according to size. The sampler was an LBL-developed flow controlled device with a 3 µm cutpoint. The pumps sampled only during hours the building was occupied. If filters had to be changed due to excessive loading, the combined weight of all filters from one site was determined—thus all values are approximately

10 working-day (80-h) averages. Buildings had varied types of smoking policies, from relatively unrestricted to very tightly controlled, as in one elementary school. In most buildings, an attempt was made to site at least one monitor in an area where smoking was allowed. Data were obtained from smoking areas in about 30 of the 38 buildings.

Results comparing smoking and non-smoking areas are provided in Table 7-17 and Figure 7-17. Mean RSP concentrations in the smoking areas were more than three times higher than in the non-smoking areas (70 versus 19 $\mu g/m^3$). Since these arithmetic means showed evidence of being driven by one or two high values, the geometric mean (averaged across all sites in a building) may be a better comparison. Here the ratio is very close to 3 to 1 (44 versus 15 $\mu g/m^3$). Outdoor results at 30 sites had the identical arithmetic mean as the indoor non-smoking sites: 18.9 $\mu g/m^3$.

Repace and Lowrey (1980) sampled 19 establishments allowing smoking (seven restaurants, three bars, church bingo games, etc.) and 14 where no smoking occurred (including five residences and four restaurants) between March and early May of 1978. Sampling occurred for short periods of time (2 to 50 min) using a TSI Piezobalance to measure $PM_{3.5}$. Indoor concentrations ranged from 24 to 55 $\mu g/m^3$ in the areas without smoking, and from 86 to 697 $\mu g/m^3$ in places with active smoking.

Miesner et al. (1989) sampled particles and nicotine in 57 locations within 21 indoor sites in Metropolitan Boston, MA, between July 1987 and February 1988. $PM_{2.5}$ was sampled using Harvard aerosol impactors. Sampling times ranged from about 3 h in a bus station to 16 h in a library, depending partly on how "clean" the environment was perceived to be. $PM_{2.5}$ concentrations ranged from 6 $\mu g/m^3$ (in the library) to 521 $\mu g/m^3$ in a smoking room in an office building. For 42 measurements in non-smoking areas, the mean $PM_{2.5}$ concentration was 25 ± 30 (SD) $\mu g/m^3$. Six of these measurements included a classroom with visible levels of chalk dust on the impactor, four measurements in subways, and the bus station. The remaining 36 nonsmoking areas had a mean $PM_{2.5}$ concentration of 15 ± 7 $\mu g/m^3$. The 15 smoking areas ranged from 20 to 520 $\mu g/m^3$ with a mean of 110 ± 120 $\mu g/m^3$.

Sheldon et al. (1988a,b) reported on the EPA 10-building study of hospitals, homes for the elderly, schools, and office buildings. Particle measurements were taken in six buildings using a National Bureau of Standards portable particle sampler (McKenzie et al., 1982) to

TABLE 7-17. SMOKING, NONSMOKING, AND OUTDOOR RSP CONCENTRATIONS AND RATIOS

Building No.	Outdoor ($\mu g/m^3$)	Indoor ($\mu g/m^3$) Arithmetic Mean (Range)			Ratios		
		Nonsmoking	Smoking[c]	Mean[d]	Indoor Nonsmoking ÷ Outdoor	Indoor Smoking ÷ Outdoor	Indoor Mean ÷ Outdoor
1	ND	25(19-36)	ND	25(19-36)	NA	NA	NA
2	ND	19(18-21)	ND	19(18-21)	NA	NA	NA
3	ND	ND	20(16-25)	20(16-25)	NA	NA	NA
4	8	7(6-8)	ND	7(6-8)	0.9	NA	0.9
5	BD	13(13)	14(14)	13(13-14)	NA	NA	NA
6	35	12(11-13)	35(23-59)	28(11-59)	0.3	1.0	0.8
7	35	38(32-44)	39(39)	38(32-44)	1.1	1.1	1.1
8	8	7(7-8)	ND	7(7-8)	0.9	NA	0.9
9	8	11(11)	16(13-20)	15(11-20)	1.3	2.0	1.9
10	9	65(53-74)	95(67-127)	86(53-127)	7.0	11.0	9.6
11[a]	8	23(9-49)	209(209)	63(9-209)	2.9	26.1	7.9
12	ND	10(10)	63(63)	36(10-63)	NA	NA	NA
13	10	5(5-6)	ND	5(5-6)	0.5	NA	0.5
14	6	ND	30(26-34)	30(26-34)	NA	5.0	5.0
15	BD	11(7-14)	12(12)	11(7-14)	NA	NA	NA
16	10	9(8-11)	73(73)	31(8-73)	0.9	7.3	3.1
17[b]	7	11(10-13)	105(105)	40(10-105)	1.6	15.0	6.1
18	7	ND	19(19)	19(19)	NA	2.7	2.7
19	7	ND	20(11-29)	20(11-29)	NA	2.9	2.9
20	18	11(10-11)	ND	11(10-11)	0.6	NA	0.6
21	17	11(9-12)	ND	11(9-12)	0.7	NA	0.7
22	20	18(18)	57(22-165)	50(18-165)	0.9	2.9	2.5
23	11	9(BD-20)	ND	9(BD-20)	0.8	NA	0.8
24	11	44(10-77)	24(24)	37(10-77)	4.0	2.2	3.4
25	68	35(32-38)	109(109)	60(32-109)	0.5	1.6	0.9
26	32	45(20-70)	82(55-123)	67(20-123)	1.4	2.6	2.1
27	52	36(33-38)	61(33-89)	48(33-89)	0.7	1.2	0.9
28	65	36(29-43)	BD	24(BD-43)	0.6	NA	0.4
29	29	10(8-12)	144(144)	32(8-144)	0.3	5.0	1.1
30[b]	33	24(20-30)	113(113	37(20-113)	0.7	3.4	1.1
31	13	12(8-18)	268(268)	64(8-268)	0.9	20.6	4.9
32	ND	13(10-17)	36(21-52)	21(10-52)	NA	NA	NA
33	ND	ND	29(12-74)	29(12-74)	NA	NA	NA
34	16	13(10-16)	54(13-117)	28(10-117)	0.8	3.4	1.8
35	18	20(6-35)	50(50)	23(6-50)	1.1	2.8	1.3
36[a]	20	14(9-18)	72(17-127)	28(9-127)	0.7	3.6	1.4
37	19	21(12-32)	27(11-62)	25(11-62)	1.1	1.4	1.3
38	14	7(BD-9)	308(308)	46(BD-308)	0.5	22.0	3.3
39	11	8(8-9)	13(11-14)	11(8-14)	0.7	1.3	1.0
40	11	10(8-12)	26(11-40)	15(8-40)	0.9	2.4	1.4
AM	19	19	70	30	1.2	6.0	2.3
ASD	16	14	73	19	1.3	7.2	2.2
GM	14	15	44	24	0.9	3.6	1.7
GSD	2.2	1.9	2.7	2.0	2.0	2.6	2.3

[a]Repeated test of building #11.
[b]Repeated test of building #17.
[c]Smoking within 10 m radius of site.
[d]Arithmetic average of all sites in building.

NA = Not applicable.
ND = No data collected.
BD = Below detection limit.

Source: Turk et al. (1987).

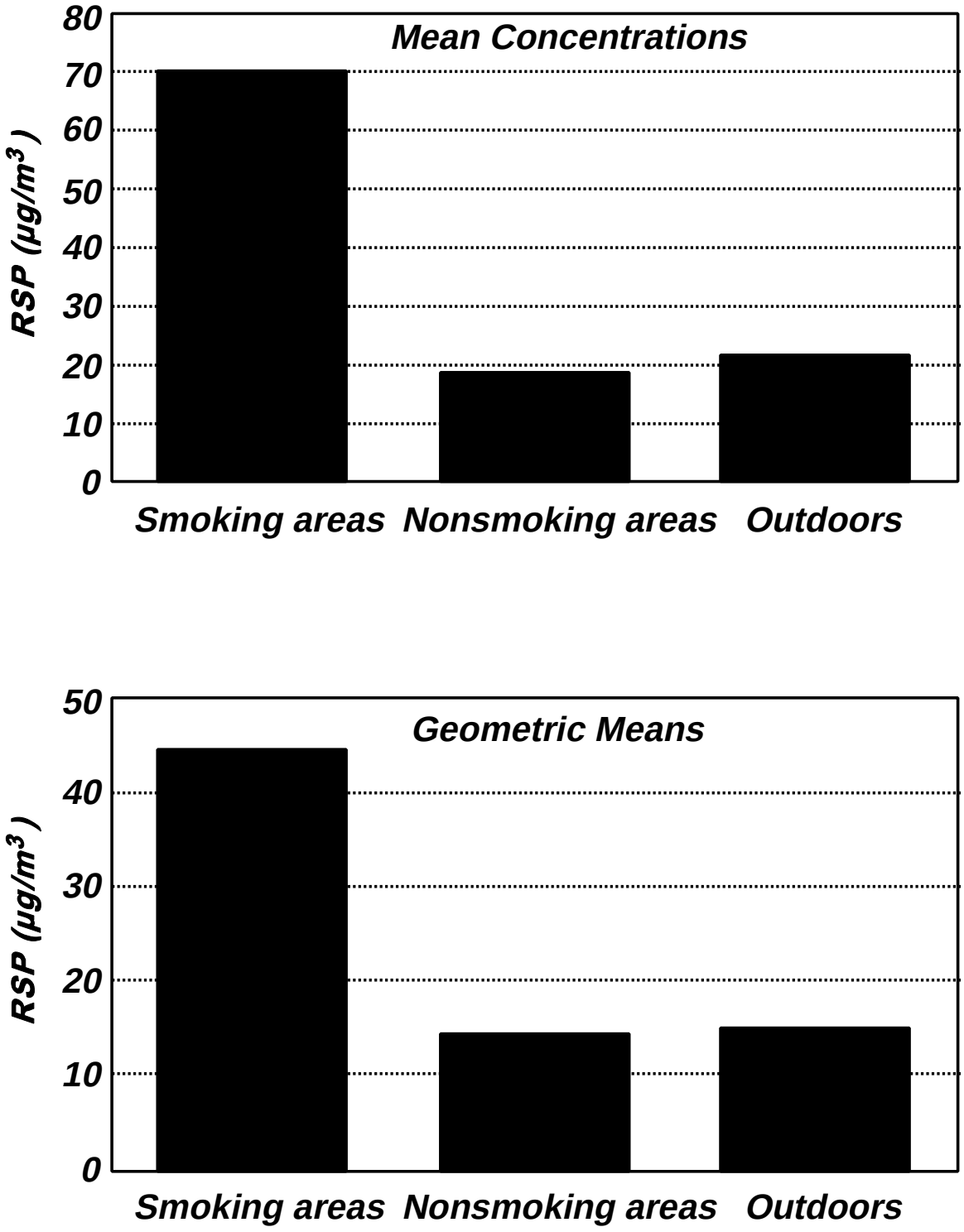


Figure 7-17. Comparison of respirable particles in smoking and nonsmoking areas of 38 buildings in the Pacific Northwest.

Source: Turk et al. (1987).

collect two size fractions: PM_3 and a coarse fraction between PM_3 and PM_{15}. The sampler employed two filters in series: an 8.0 μm Nuclepore filter for PM_{15} and a 3 μm Ghia Zefluor Teflon filter for fine particles. The flow rate was 6 Lpm for a 24-h sample. Three consecutive 24-h samples were collected at each building. Additional particle monitoring was provided at certain locations (e.g., smoking lounge, cafeteria) using a Piezobalance ($PM_{3.5}$) and a dichotomous sampler ($PM_{2.5}$ and PM_{10}).

In areas without smoking, indoor concentrations of both size fractions were generally lower than outdoor levels; for example, the coarse fraction ranged from 0.2 to 0.66 of the outdoor level (13 to 17 μg/m^3) in the three buildings with no smoking. The fine fraction was present at higher indoor-outdoor ratios, ranging from 0.56 to 0.99 in the same three buildings (outdoor fine fraction ranged from 16 to 33 μg/m^3). The fine fraction was elevated in the regions of smoking (range of 14 to 56 μg/m^3). Piezobalance results for several buildings showed uniformly low (7 to 29 μg/m^3) for 800 min of monitoring in nonsmoking areas.

Concentrations in the areas allowing smoking were more often in the 40 to 60 μg/m^3 range, with short-term peaks as high as 345 μg/m^3. It was possible to use the observed declines in $PM_{3.5}$ following cessation of smoking to calculate an effective air exchange rate and thus a source strength for $PM_{3.5}$ emissions from cigarettes. Four estimates gave an average value of about 6 mg/cigarette, somewhat below the chamber study estimates of 10 to 15 mg/cig. An estimate due to Repace and Lowrey (1980) of concentrations of respirable particulates due to smoking was also tested, with good agreement. The Repace and Lowrey equation is

$$C\ \mu g/m^3 = 27.6\ P_a/a \qquad (7\text{-}7)$$

where P_a is smoking occupancy in persons per 100 square meters and a is the air exchange rate h^{-1}. Equation 7-7 was developed assuming one of every three occupants are smokers who smoke two cigarettes per hour. Assuming a background concentration of 15 μg/m^3, the measured values for the smoking lounge for zero, three, and nine smokers were 10, 78, and 284 μg/m^3, respectively. Equation 7-7 predicts 0, 99, and 296 μg/m^3, respectively. In two of the homes for the elderly, apartments with smokers and nonsmokers were measured for three consecutive days using the NBS samplers. In one building, the smoker's apartment had a 2-day PM_3 average of 39 μg/m^3, compared to 9.4 μg/m^3 in the nonsmoker's apartment; in the other home for the elderly,

where two smokers shared one apartment, the average 2-day PM_3 concentration was 88 µg/m^3 compared to 8.6 µg/m^3 in the nonsmoking apartment. The simultaneous ambient values were not measured at Home 1. At Home 2, the ambient value was 11 μg/m^3.

Owen et al. (1990) studied particle size distributions in an office under varying conditions of ventilation and occupancy. The unoccupied office using minimum outdoor air had concentrations at least as low as the occupied office using maximum outdoor air. $PM_{3.5}$ concentrations (measured using the TSI Piezobalance) were about twice as high (75 versus 39 μg/m^3) in the occupied office when the dampers were closed as when they were open. The main source of particle generation appeared to be the hallway, suggesting that resuspension of tracked-in dust was an important indoor source of particles as reported by Roberts et al. (1990) for residences.

7.2.3 Indoor Air Quality Models and Supporting Experiments

Indoor concentrations of particles are a function of penetration of outdoor particles and generation of particles indoors. The concentrations are modified by air exchange rates and deposition rates of the particles onto indoor surfaces.

7.2.3.1 Mass Balance Models

Mass balance models have been used for more than a century in various branches of science. All such models depend on the law of the conservation of mass. They simply state that the change in mass of a substance in a given volume is equal to the amount of mass entering that volume minus the amount leaving the volume. Usually they are written in the form of first-order linear differential equations. That is, consider a volume V filled with a gas of mass m. The change in mass Δm over a small time Δt will simply be the difference between the mass entering the volume (m_{in}) and the mass leaving the volume (m_{out}):

$$\frac{\Delta m}{\Delta t} = \frac{(m_{in} - m_{out})}{\Delta t} \qquad (7\text{-}8)$$

Taking the limit as Δt approaches zero, we have the differential equation for the rate of change of the mass:

$$\frac{dm}{dt} = \frac{d}{dt}\,(m_{in} - m_{out}) \qquad (7\text{-}9)$$

If we require that the mass be uniformly distributed throughout the volume at all times, we have a condition that the physical chemists call "well-mixed". We assume that any mass gained or lost in the volume V is instantaneously distributed evenly throughout the volume. We may then replace the mass term (m) by the concentration $C = m/V$, so that $dm/dt = V\,dC/dt$.

The above equations are the basis for all such mass-balance models. Equation 7-9 takes on many forms depending on the type of processes involved in transporting mass into or out of the volume being considered. A large class of models assume that the volume V is a single perfectly mixed compartment. More complex models assume multiple compartments to allow for incomplete mixing in the total volume V (Mage and Ott, 1996). A detailed mass-balance model that includes changes in particle size, chemical composition, and turbulence is described in Nazaroff and Cass (1989).

7.2.4 Summary of Indoor Particulate Matter Studies

At low outdoor levels of fine ($PM_{3.5}$ or $PM_{2.5}$) particles (as in most of the cities in the Harvard Six-City and New York State studies), mean indoor concentrations have been found to be twice as high as outdoor levels. However, for homes without smokers or combustion sources, indoor levels are often roughly equal to outdoor levels (Santanam et al., 1990; Leaderer et al., 1994; Neas et al., 1994). At high outdoor levels, mean indoor concentrations have been about 10% lower than the mean outdoor concentrations in the two areas studied (Steubenville, OH, and Riverside, CA). Indoor concentrations are considerably higher during the day, when people are active, than at night. Based on a mass-balance model, outdoor air was the major source of indoor particles in the PTEAM study, providing about 3/4 of fine particles ($PM_{2.5}$) and 2/3 of inhalable particles (PM_{10}) in the average home. However, outdoor air contributed less than half of the indoor particle concentrations at seven out of eight other sites with extensive indoor-outdoor measurements. Indoor concentrations are much higher during the day, when people are active, than at night.

In the PTEAM study (with very high outdoor particle concentrations), indoor levels were significantly influenced by outdoor levels, but with relatively low R^2 values ranging between

0.22 and 0.54. In the other two major studies, no significant indoor-outdoor relation was observed. Regressions of indoor on outdoor particles seldom explained more than half the variance of any study ($R^2 < 50\%$). However, in those studies with repeated measures on the same house, (e.g., the PTEAM prepilot [Table 7-6], the Phillipsburg, NJ, study [Table 7-15] and Tamura et al. [1996] in Section 7.4.2.1), longitudinal regressions of indoor on outdoor particles often had much higher R^2 values of 0.6 to 0.9 for each individual house. Since the epidemiological studies of health effects of particles have been studies of variation over time, the longitudinal regressions by individual home are expected to be more relevant to the epidemiology studies than cross-sectional regressions across all homes in the study. The better relationship showed by these regressions suggests that whatever structural or behavioral characteristics affect indoor particle concentrations in the home tend to persist or be repeated over time. This gives better support to the epidemiological findings than would be inferred from the typically low R^2 values reported for the cross-sectional regressions performed in most studies.

Deposition rates k ranged from 0.16 h^{-1} for sulfur to 0.4 h^{-1} for fine ($PM_{2.5}$) particles to 1 h^{-1} for coarse particles ($PM_{10} - PM_{2.5}$), with an intermediate estimate of 0.65 h^{-1} for PM_{10}. The penetration factor P for both fine and coarse fractions was estimated to be unity. For a home with no indoor sources whatever and a typical air exchange rate of about 0.75 h^{-1}, these values for k and P would imply that sulfur indoors would be about 0.75/(0.16 + 0.75) = 82% of the outdoor value at equilibrium, fine particles indoors would be about 0.75/(0.4+0.75) = 65% of the outdoor value at equilibrium, indoor PM_{10} would be about 54% of outdoor levels, and indoor coarse particles would be about 43% of outdoor levels. Since very few homes were observed to have concentrations this low, it can be inferred that very few homes are free of important indoor sources of particles.

A crucial question is the impact of outdoor particles on indoor particle concentrations. It was found that the governing equation is a function of only two parameters: air exchange rate a and particle deposition rate k: $a/(a+k)$. Air exchange rates measured in the United States appear to follow a roughly log-normal distribution with a geometric mean of 0.5 and a geometric standard deviation about 2. With the values for the deposition rates provided above, one can calculate the impact of outdoor particles on indoor concentrations for any given value of the air exchange rate. At a low air exchange rate of, say, 0.4 h^{-1}, sulfates indoors will be 71% of their

outdoor values, fine particles indoors will be 50% of their outdoor values, while coarse particles will be 0.4/1.4 or 28% of their outdoor values. At a higher air exchange rate of 1 h^{-1}, sulfates will be 86% of their outdoor concentration, fine particles will be 1/1.4 or 71% of their outdoor concentration, whereas coarse particles will be 50% the outdoor concentration. The difference in both cases between the two size fractions is about 0.2; that is, for the entire range of realistic air exchange rates (from 0.2 h^{-1} to 2 h^{-1}), if the fraction of outdoor coarse (PM_{10} – $PM_{2.5}$) particles found indoors is *f*, then the fraction of fine particles found indoors will be approximately $f + 0.2$. It can be seen that a reduction in air exchange rate would reduce the impact of outdoor air on indoor air particle concentrations.

7.2.5 Bioaerosols

Biologically-derived particles are frequently ignored components of both ambient and indoor aerosols. This lack of attention is, in part, due to the fact that the bioaerosols are considered "natural" and not amenable to control. Methods for their analysis are, in many cases, highly variable, and very little exposure or exposure/response information is available. Measurement methods for bioaerosols are discussed in Chapter 4 (Section 4.4). Various health effects associated with bioaerosols are discussed in Chapter 11. A few reference works that focus on bioaerosols include Gregory (1973), Edmonds (1979), Cox (1987), Lighthart and Mohr (1994), and Cox and Wathes (1995).

For bioaerosols, there is considerable confusion among the terms reservoir, source, particle, and agent. For the purposes of this chapter, the following definitions apply:

- Reservoir: the environmental niche in which source organisms are living
- Source: the organism that produced the particle
- Particle: the particle shed from the organism
- Agent: the part(s) of the particle that actually mediate the disease process.

Examples of bioaerosol sources, particles and agents are presented in Table 7-18.

TABLE 7-18. AN OVERVIEW OF ORGANISMS, AEROSOLS, AND DISEASE AGENTS

Sources	Aerosol Particles	Disease Agents
Plants	Pollen and pollen fragments, fragments of other plant parts, spores (ferns, mosses), algal cells	Glycoprotein allergens
Animals	Skin scales, secretions (saliva, skin secretions), excreta, body parts (arthropods)	Glycoprotein allergens
Fungi	Spores, hyphae, yeast cells, metabolites (toxins, digested substrate material)	Glycoprotein allergens, infectious units, glucans, mycotoxins
Bacteria	Cells, fragments, metabolites (toxins, digested substrate material)	Infectious units, allergens, endotoxin, exotoxins
Viruses	Viral particles	Infectious units

7.2.5.1 Plant Aerosols

Pollen

Pollen is produced by vascular flowering plants: trees (pines, cedars, birch, elm, maple, oak, hickory, walnut, etc.), grasses, and weeds (ragweed, sage, Russian thistle, lambs quarters, etc.). Within these large groupings, specific types are regionally common. For example, ragweed is most common in the eastern United States. Birch pollen dominants the spring pollen season in New England, while mountain cedar pollen is abundant early in the year in the southwest (Lewis et al., 1983).

Pollen levels outdoors are controlled by the number of plants available for pollen release, the amount of pollen produced by each plant, factors that control pollen release and dispersion from the plant, and factors that directly affect the aerosols (Edmonds, 1979). The number of plants available depends on the many environmental factors that control plant prevalence, some of which are human factors. As an example, the abundance of the ragweed plant in a particular year depends on the number of plants that produced seed in the previous year, disturbed ground available for seed germination and growth, and meteorological factors during the growing season. Once a crop of ragweed has been produced, pollen production depends on temperature, rainfall, and day length.

Pollen grains are relatively large complex particles that consist of cellular material surrounded by a cell membrane and a complex wall. Pollen grain structure has been well studied. Pollen shed is controlled by temperature, humidity, wind, and rain. Pollen levels in air depend on all of these factors as well as wind and rain conditions after release, and on surfaces available for impaction. Figure 7-18 represents day to day ragweed pollen prevalence in Kalamazoo, MI, for 1994.

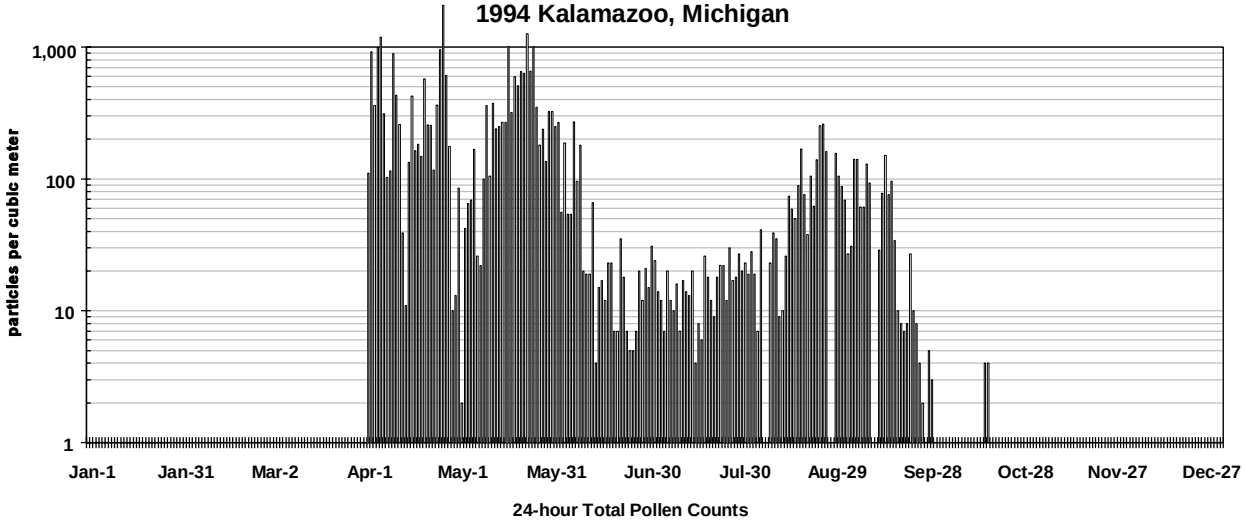


Figure 7-18. Chart of ragwood pollen prevalence. Sampling was not conducted before April and during the first few days of October.

Source:

Pollen allergens are (apparently) water-soluble glycoproteins that rapidly diffuse from the grain when it contacts a wet surface. The glycoproteins are (generally) specific to the type of pollen, although large groups may be represented by a single allergen. For example, many different kinds of grasses carry similar allergens in their pollen grains. A number of pollen allergens have been characterized: Amb a I (ragweed), Bet p I (birch), Par j I (parietaria), etc.

Other Natural Plant Aerosols

Other plant-derived particles that are a natural part of outdoor air include algal cells; spores of mosses, liverworts, club mosses, and ferns; and fragments of all kinds of plants. Very little has been reported about the prevalence or human impact of any of these aerosol particles, although they are presumed to carry allergens.

Man-Made Plant Aerosols (Soy, Latex, Occupational)

Man-made accumulations of plant material that are subsequently handled inevitably produce bioaerosols. The most common practices that involve such accumulations are storage, handling, and transport of farm products (hay, straw, grain), composting, and manufacturing processes that involve the use of plant material. In addition, the use of some plant products can result in disease-causing aerosols (Alberts and Brooks, 1992). The aerosols produced from most of these processes are complex, and few have been accurately characterized.

Grain Dust. It is well-recognized that grain dusts include respirable-size particles (< 10 μm) although the exact nature of the particles and the agents of disease remain speculative. Soybean dust aerosols released from freighters unloading the beans in port have been blamed for epidemics of asthma.

Wood Dust. Wood trimmer's disease (from particles released from wood during high-speed cutting). Sewage composting involves the use of wood chips that can release allergenic aerosols.

Latex. Latex-containing powder aerosols are produced when surgical gloves are used. Latex particles also may be released from automobile tires.

7.2.5.2 Animal Aerosols

Mammalian Aerosols

All mammals produce aerosols, from humans to the smallest mouse. Human aerosols (skin scales, respiratory secretions) do not cause disease except, of course, for agents of infection (see below). Other mammals release aerosols that cause hypersensitivity diseases. The most common of these are cats, dogs, farm animals, laboratory animals, and house mice, although all animals release aerosols that could be sensitizing under appropriate conditions (Burge, 1995). Mammals only cause human disease when appropriate exposure conditions occur. For cats, simply having a cat in a house will create such conditions, as will handling any animal regardless of the environment. Cat allergens apparently become aerosolized on very small particles (<1 μm) shed from skin and saliva. There is some indication that dog, mouse, and other rodent allergens are borne on dried urine particles, and particle sizes are similar to those of cat allergen. Little is known about other mammalian aerosols. Cat and dog allergens have been characterized (Fel d I, Can f I) and other mammalian allergens are under active study.

Avian Aerosols

Wild and domesticated birds associated with disease-causing aerosols include for example: starlings (histoplasmosis); pigeons (histoplasmosis, pigeon-breeders disease); parrots (psittacosis); poultry (poultry-handlers disease); etc. Of these diseases, only the hypersensitivity diseases (pigeon breeders and poultry handlers disease) are caused by "bird" aerosols. The others are infections caused by agents inhabiting the birds (see below). The birds that release antigens that have caused human disease are those that are confined or congregate close to people. The avian aerosol-hypersensitivity diseases are almost exclusively confined to sites where birds are bred and handled extensively, especially in indoor environments. Relatively little is known about avian aerosols. Probably skin scales, feather particles, and fecal material are all released as antigen-containing aerosols. The antigens (allergens) responsible for avian-related diseases have not been characterized.

Insect Aerosols

Dust Mites. Dust mites are arthropods belonging to the family Pyrogliphidae. There are two common species in temperate climates: *Dermatophagoides farinae* (which proliferates under relatively dry conditions) and *D. pteronyssinus* which dominates populations in more humid environments (Arlian, 1989). Dust mites thrive in environments where relative humidity consistently exceeds 60 % and where skin scales and fungal spores are available as a food source. Primary reservoirs for exposure are bedding and carpet dust. The mite itself is about 100 μm long, but it excretes 20 μm membrane-bound fecal particles that contain the allergens. Exposure to dust mite allergens apparently occurs only when reservoirs are disturbed. Dust mites produce allergens that are a major cause of sensitization in children. The allergens are digestive enzymes that gradually diffuse from fecal particles after deposition on mucous membranes. Several dust mite allergens have been characterized and monoclonal antibodies against each have been raised and cloned. These include Der f I and II, and Der p I and II (Platts-Mills and Chapman, 1987).

Cockroaches. Cockroaches are insects belonging to the Orthoptera (Mathews, 1989). The most common cockroach infesting temperate climate buildings is *Blatella germanica*, the German cockroach. Cockroaches are nocturnal, and inhabit dark environments where food and water are available. Common food sources include stored animal or human food, and discarded food (garbage). Cockroaches are extremely prolific, given appropriate environmental conditions. Population pressure will eventually drive the roaches into the daylight in search of food. Cockroaches shed body parts, egg cases, and fecal particles, all of which probably carry allergens. Little is known about the particles that actually carry the allergens. Two German cockroach allergens have been characterized: Bla g I, and Bla g II. The function within the cockroach of these allergens is unknown. Cockroach allergens are probably a major cause of asthma for some populations of children.

Other Insects. Fragments of gypsy moths and other insects that undergo massive migrations can become abundant in ambient air. Sizes, nature, and allergen content of such particles have not been studied. Cases of occupational asthma from exposure to insects (e.g., sewer flies) have been reported.

Other Animal Allergens

It is likely that proteinaceous particles shed from any animal could cause sensitization if exposure conditions are appropriate. For example, exposure to proteins aerosolized during seafood processing have caused epidemics of asthma.

7.2.5.3 Fungal Aerosols

Fungi are primarily filamentous microorganisms that reproduce and colonize new environments by means of airborne spores. Most use complex non-living organic material for food, require oxygen, and have temperature optima within the human comfort range. The major structural component of the cell wall is acetyl-glucosamine polymers (chitin). Cell walls also may contain B-glucans, waxes, mucopolysaccharides, and a wide variety of other substances. In the process of degrading organic material, the fungi produce CO_2, ethanol, many other volatile organic compounds, water, organic acids, ergosterol, and a broad spectrum of secondary metabolites including many antibiotics and mycotoxins.

The fungi colonize dead organic materials in both outdoor and indoor environments. Some fungi are able to invade living plant tissue and cause many important plant diseases. A few fungi will invade living animal hosts, including people. Fungi are also universally present in indoor environments unless specific efforts are made for their exclusion (i.e., as in clean rooms). The kinds of fungi that are able to colonize indoor materials are generally those with broad nutritional requirements (e.g., *Cladosporium sphaerospermum*), those that are able to colonize very dry environments (e.g., members of the *Aspergillus glaucus* group), or organisms that readily degrade the cellulose and lignin present in many indoor materials (e.g., *Chaetomium globosum*, *Stachybotrys atra*, *Merulius lacrymans*). Yeasts (which are unicellular fungi) and other hydrophilic taxa (e.g., Fusarium, Phialophora) are able to colonize air/water interfaces. Water, in fact, is the most important factor controlling indoor fungal growth, since food sources are ubiquitous (Kendrick, 1992).

Particles that become airborne from fungal growth include spores (the unit of most fungal exposure), fragments of the filamentous body of the fungus, and fragments of decomposed substrate material. Fungal spores range from about 1.5 μm to >100 μm in size and come in many different shapes. The simplest are smooth spheres; the most complex are large multicellular branching structures. Most fungal spores are near unit density or less. Some

include large air-filled vacuoles. Fungal spores form the largest and most consistently present component of the outdoor bioaerosols. Levels vary seasonally, with lowest levels occurring during periods of snow. While rain may initially wash large dry spores from the air, these are immediately replaced by wet (hydrophilic) spores that are released in response to the rain.

Some kinds of spores are cosmopolitan in outdoor air (e.g., *Cladosporium herbarum*, *Alternaria tenuissima*). Others produced by fungi with more fastidious nutritional requirements are only locally abundant. A typical indoor fungal aerosol is composed of particles penetrating from outdoors, particles released from active growth on indoor substrates, and reaerosolized particles that have settled into dust reservoirs. Indoor fungal aerosols are produced by active forcible discharge of spores, by mechanisms intrinsic to the fungus that "shake" spores from the growth surface, and (most commonly) by mechanical disturbance (e.g., air movement, vibration).

Allergic rhinitis and asthma are the only commonly reported diseases resulting from fungal exposures outdoors, and which also commonly occur indoors. The allergens of fungi are probably digestive enzymes that are released as the spore germinates. Other spore components (of unknown function) may also be allergenic. Only very few fungal allergens (out of possibly hundreds of thousands) have been characterized: (e.g., *Alt a* I, *Cla h* I, and *Asp f* I).

Allergic fungal sinusitis and allergic bronchopulmonary mycoses occur when fungi colonize thick mucous in the sinuses or lungs of allergic people. The patterns of incidence of allergic fungal sinusitis may be explained in part by geographic variability in ambient fungal exposures. Figure 7-19 shows total fungal spore counts in Kalamazoo, MI, for 1994. This disease is most commonly caused by Bispora, Curvularia, and other dark-spored fungi. Exposure patterns required for allergic bronchopulmonary mycoses are unknown. This disease is usually caused by *Aspergillus fumigatus.* Histoplasmosis and Coccidioidomycoses are fungal infectious diseases that result from outdoor exposures to *Histoplasma capsulatum* (a fungus that contaminates damp soil enriched with bird droppings) and *Coccidioides inmitis* (a fungus that growth in desert soils. Indoor aerosol-acquired fungal infections are rare, and restricted to immunocompromised people (Rippon, 1988).

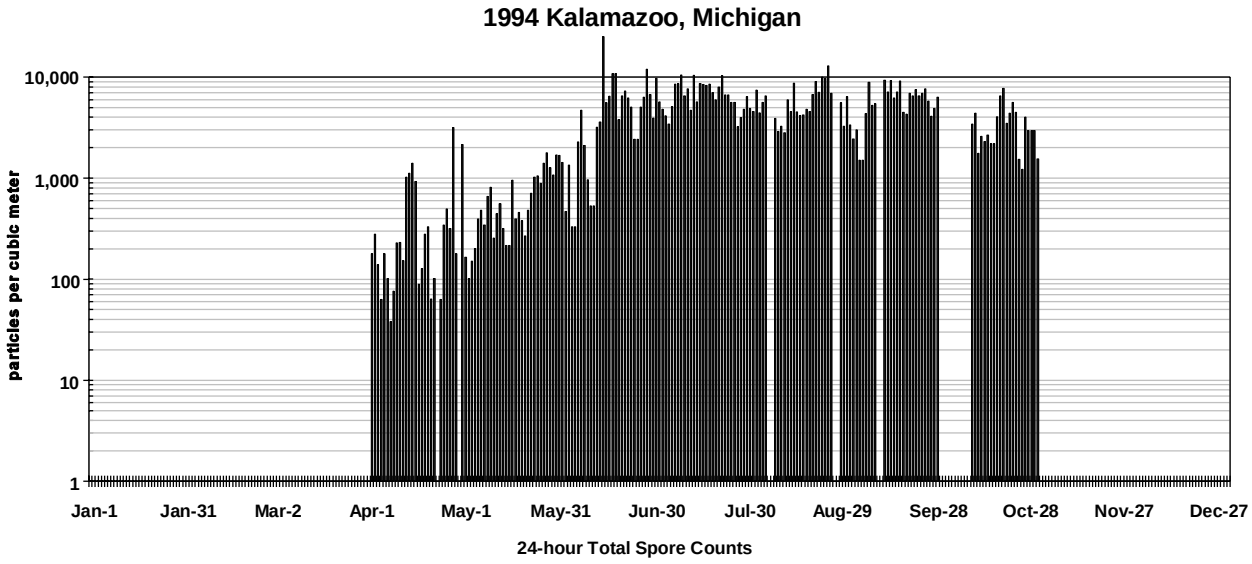


Figure 7-19. Chart of fungal spore prevalence in Kalamazoo, MI, for 1994.

Source:

Toxic agents produced by fungi include antibiotics, mycotoxins, and some cell-wall components that have toxic or irritant properties. The antibiotics and mycotoxins are secondary metabolites that are produced during fungal digestion of substrate materials, and their presence depends, in part, on the nature of the substrate. The locations of the toxins in spores or other mycelial fragments are unknown, as are the dynamics of release in the respiratory tract. Aerosol exposure to fungal antibiotics in levels sufficient to cause disease is unlikely. Mycotoxicoses have been reported as case studies from exposure to spores of *Stachybotrys atra* (Croft et al., 1986), and epidemiologically for *Aspergillus flavus* (Baxter et al., 1981).

7.2.5.4 Bacterial Aerosols

Bacteria, in contrast to plants, animals and fungi, contain neither nuclei or mitochondria. Most are unicellular, although some form “pseudo” filaments when cells remain attached following cell division. The actinomycetes are bacteria that do form filaments and (in some cases) dry spores designed for aerosol dispersal. The bacteria can be broadly categorized into two groups based on a response to the Gram stain procedure. The cell walls of Gram positive bacteria are able to absorb a purple stain; the walls of Gram negative bacteria resist staining. The Gram negative cell wall contains endotoxin (see above).

Most infectious agents are maintained in diseased hosts. A few, including *Legionella pneumophila*, reside in water-filled environmental reservoirs such as water delivery systems, cooling towers, air conditioners, and (outdoors) oceans, lakes, streams, etc.

Infectious agents are often released from hosts in droplets released from the respiratory tract. Each droplet contains one or more of the infectious agent, probably one or more other organisms, and respiratory secretions. Most droplets are very large and fall quickly. Smaller droplets dry quick to droplet nuclei, which range in size from the size of the individual organism (<1 μm for the smallest bacteria) to clumps of larger organisms (>10 μm for larger bacteria). Environmental-source aerosols are produced by mechanical disturbances that include wind, rain splash, wave action, and by mechanical disturbance such as occurs in recirculation and sprays of washes and coolants, and in humidifiers. Particle sizes from all of these activity cover a wide range from well below 1 μm to >50 μm. The thermophilic actinomycetes produce dry aerial spores that require only slight air movements to stimulate release. Each spore is about 1 μm in diameter.

Whole living bacteria are agents of infectious disease (e.g., Tuberculosis, Legionnaires' disease). For tuberculosis, a single virulent bacterial cell deposited in the appropriate part of the lung is likely to cause disease in a host without specific immunity. For Legionnaires' disease, the number of organisms required to make disease development likely depends on how well the host's general protective immune system is operating. Some bacteria release antigens that cause hypersensitivity pneumonitis. The antigens may be enzymes (e.g., *Bacillus subtilis* enzymes used in the detergent industry) or may be cell wall components as in the thermophilic actinomycetes. Bacteria also produce toxins of which endotoxin is the most important from an aerosol exposure point of view.

7.2.5.5 Viral Aerosols

The viruses are units of either RNA or DNA surrounded by a protein coat. They have no intrinsic mechanism for reproduction, and require living cells whose enzyme systems they utilize to make new particles. They can be crystallized and remain able to reproduce, and are often considered intermediates between non-life and life. Because viruses require living cells to reproduce, reservoirs for them are almost exclusively living organisms. Rarely, viruses survive (but do not reproduce) in environmental reservoirs from which they are re-aerosolized to cause

disease. The Hanta virus that causes severe respiratory disease in people exposed to intense aerosols of infected mouse urine is an example of this phenomenon. Viral aerosols are produced when the infected organism coughs, sneezes, or otherwise forces respiratory or other secretions into the air. The viral particles are coated with secretions from the host, and, as for the bacteria, there may be one to many in a single droplet. The size of a single viral particle is very small (a small fraction of a μm). However, infectious droplets are probably within a much larger size range (1 to 10 μm). Each kind of virus produces a specific disease, although some of the diseases present with similar symptoms. Thus, the measles virus produces measles, the chicken pox viruses produces chicken pox and shingles. Influenza and common colds are produced by a range of viruses all of which produce symptoms that are similar (but not necessarily identical).

7.2.5.6 Ambient and Indoor Air Concentrations of Bioaerosols

A general rough estimate of the contribution of bioaerosols to collected PM mass can be made as follows: for an "average" 3 µm spherical spore of 0.9 density, each spore would weigh $\approx 13 \times 10^{-6}$ µg; for a clean indoor environment with $\approx 10^3$ spores/m^3 the mass would be on the order of 0.01 µg/m^3; for a typical outdoor condition, with $\approx 50 \times 10^3$ spores/m^3, the contribution would be on the order of 0.5 µg/m^3. In contaminated indoor environments, where spore levels above 10^6 spores/m^3 are possible, the spore weight could be on the order of 10 µg/m^3 or more.

In summary, the minor mass concentrations of bioaerosols in ambient and indoor air are independent of the concentrations of the non-bioaerosol constituents in ambient and indoor air. However, the deposition of bioaerosols at the same respiratory tract loci as the other PM can cause irritation and infection foci that may make the affected host more susceptible to the effects of other deposited PM.

7.3 DIRECT METHODS OF MEASUREMENT OF HUMAN PM EXPOSURE BY PERSONAL MONITORING

7.3.1 Personal Monitoring Artifacts

Human exposure to air pollution can be measured by placing a personal exposure monitor (PEM) close to the breathing zone of an individual. However, the very act of studying the subjects can alter their behavior, which influences the measured values of their exposures and creates an erroneous reading. This influence, known as the "Hawthorne Effect" (Mayo, 1960; Last, 1988), arises because the subjects are aware of the study objectives, and the presence of the PEM on their body is a constant reminder.

The physical location of the monitor inlet, as worn by the subject, can also influence the subject's PM exposure and the recorded PM (Cohen et al., 1982, 1984). The movements of the subject's body and the PEM sampling flow rate can alter the air currents in the subject's breathing zone. "The presence of the body and its movement affect what a personal sampler collects" (Ogden et al., 1993). When in close proximity to a source actively emitting PM (within a meter) a small change in PEM position (e.g. from left side to right side) can vary the PM measurement. The vertical position of the personal monitor sampling inlet (e.g., at the waist or at the lapel near the breathing zone), can influence the captured amount of PM that is generated from the floor and stuffed furniture (Aso et al., 1993).

In performance of a personal monitoring study, people often refuse to participate. The refusal rate increases with the burden on the respondents due to the time required to complete questionnaires, diaries and the need to carry the personal monitor with them throughout the study. If the cohort of people who refuse to participate have significantly different personal PM exposures than the participants, then the study will produce a biased estimate of the exposures of the total population.

Two other important errors that influence the personal exposure measurements are: (1) "the monitor effect", by which the monitor reduces PM concentration in the breathing zone by "self dilution" (Cohen et al., 1984), the alteration of stream lines in the area of the nose and mouth, or by electrostatic charge on a plastic cassette filter holder collecting charged particles (Cohen et al., 1982); and (2) "the subject effect", by which the subject